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American Journal of Diseases of Children

Vol. 14 AUGUST, 1917 No. 2

EPIDEMIOLOGIC DATA IN THE POLIOMYELITIS EPIDEMIC IN NEW YORK STATE*

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While it cannot be said that any really new facts have been established regarding poliomyelitis as the result of last summer's great epidemic, certain more or less widely accepted opinions concerning the periods of infectivity, the age and sex incidence and the behavior of the disease under urban and rural conditions of living have been placed on a more solid scientific footing. I shall purposely omit statistical results of serum treatment, as I do not believe it possible at present to pronounce a definite opinion as to its value.

ORIGIN, SPREAD AND MAGNITUDE OF THE EPIDEMIC

The number of cases of poliomyelitis reported in New York State during the first five months of 1916 (forty-one) was quite low; in fact, below normal, affording no intimation of the sudden flare-up that was to follow. The general invasion of the state seems to have come from the Borough of Brooklyn, where the epidemic started in the second week in June, and within a month had caused a thousand cases a week in the greater city alone, during the week ending July 16.

Chart 1, tracing the course of the epidemic week by week, shows that in the state outside New York City the disease did not assume epidemic proportions until several weeks later, and ran its course much more gradually. It spread along the routes of travel out of New York City, particularly to the suburban towns and villages on Long Island, across the river into New Jersey and northward into Westchester. It then invaded the counties bordering on the Hudson River, and along the railroad lines leading north and northwest out of the greater city. The disease often "jumped" communities in the most astounding way. Many of the local outbreaks in the Catskill counties and up the Hudson were clearly traceable to imported cases brought in from New York

* Submitted for publication May 30, 1917.

* Read by title at the annual meeting of the American Pediatric Society, White Sulphur Springs, W. Va., May, 30, 1917.

City. In the central and northern parts of the state the epidemic did not reach its height until well along in August, and did not subside until the latter part of October.

During the period from June to December, 1916, there occurred in New York State over 13,000 cases and 3,300 deaths. Of the total number, 8,991 cases, or more than two thirds, occurred in New York City, the rest of the state reporting 4,186 cases.

COMPARATIVE INCIDENCE AND FATALITY OF THE DISEASE UNDER URBAN AND RURAL CONDITIONS

Table 1, together with Chart 2, shows strikingly the difference in the prevalence and fatality of the epidemic in the different sections of the state.

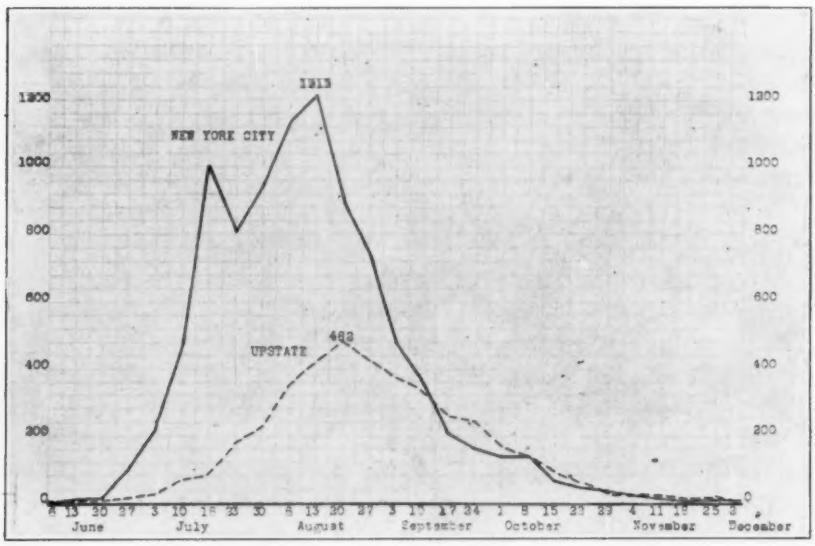

Chart 1.—Reported cases of poliomyelitis, week by week, during the epidemic of 1916 in New York City (dates of report) and upstate (dates of onset).

It will be noted that the districts affected in rural New York comprised but one half of its total population, while very few of the cities escaped the disease altogether. On the other hand, in those districts of rural New York that were affected, a much higher proportion of the population was attacked with poliomyelitis than in either New York City or the upstate cities. Thus, on the average, 2.4 persons per thousand population in the rural sections affected were attacked by the disease during the course of the epidemic, as compared with 1.6 in New York City, and 0.6 per thousand in the upstate cities as a whole.

TABLE 1.—INCIDENCE AND FATALITY FROM POLIOMYELITIS DURING THE EPIDEMICS OF 1916 AND 1912

Area	Population of Areas Affected	Number of Cases	Case Rate per 10,000 Population	Number of Deaths	Fatality Rate per 100 Cases	Death Rate per 10,000 Population
June–December, 1916 New York City.....	5,602,841	8,991	16.0	2,444	27.2	4.4
Upstate cities..... (Total Pop., 2,279,047)	2,120,538	1,300	6.1	294	22.6	1.4
Rural New York..... (Total Pop., 2,417,814)	1,223,424	2,886	23.6	572	19.8	4.7
July–October, 1916 Newark, New Jersey...	366,721	1,390	37.9	372	26.8	10.1
1912 Buffalo, New York.....	423,711	297	7.0	39	13.1	0.9
New York state..... (Cases observed by Surgeon Frost)	.....	1,108	....	183	16.5	

TABLE 2.—MOVEMENT OF CASES, DEATHS AND FATALITY RATES FROM POLIOMYELITIS DURING THE EPIDEMIC OF 1916 IN NEW YORK STATE, BY MONTHS

Month	State of New York			New York City			Rest of State		
	Cases	Deaths	Fatality Rate per 100 Cases	Cases	Deaths	Fatality Rate per 100 Cases	Cases	Deaths	Fatality Rate per 100 Cases
June.....	367	64	17.4	313	63	20.1	54	1	
July.....	4,011	895	22.3	3,443	779	22.6	568	116	20.4
August....	5,987	1,466	24.5	3,927	1,080	27.5	2,060	368	17.9
September.	1,992	628	31.5	985	364	37.0	1,007	264	26.2
October....	645	215	33.3	258	122	47.3	387	93	24.0
November.	135	40	29.6	47	25	53.2	88	15	17.0
December..	40	20	50.0	18	11	61.1	22	9	40.9
Total....	13,177	3,310	25.1	8,991	2,444	27.2	4,186	866	21.1

Table 2 shows the movement of cases, deaths and fatality rates by months, from June through December. Of the 13,177 cases reported in New York State during that period, 3,310 patients died, indicating a fatality rate of about 25 per cent., or one death out of every four cases, which is much higher than that observed in any considerable epidemic hitherto recorded. As between the different sections, it is notable that in the rural sections a much lower proportion of the cases proved fatal than in the cities. From Chart 2 (darkened portions) it

will be seen that 19.8 per cent. of the rural cases ended in death, the upstate cities following with 22.6 deaths per one hundred cases, and the New York City cases showing a decidedly higher proportion of fatalities than either of the other two areas — 27.2 per cent.

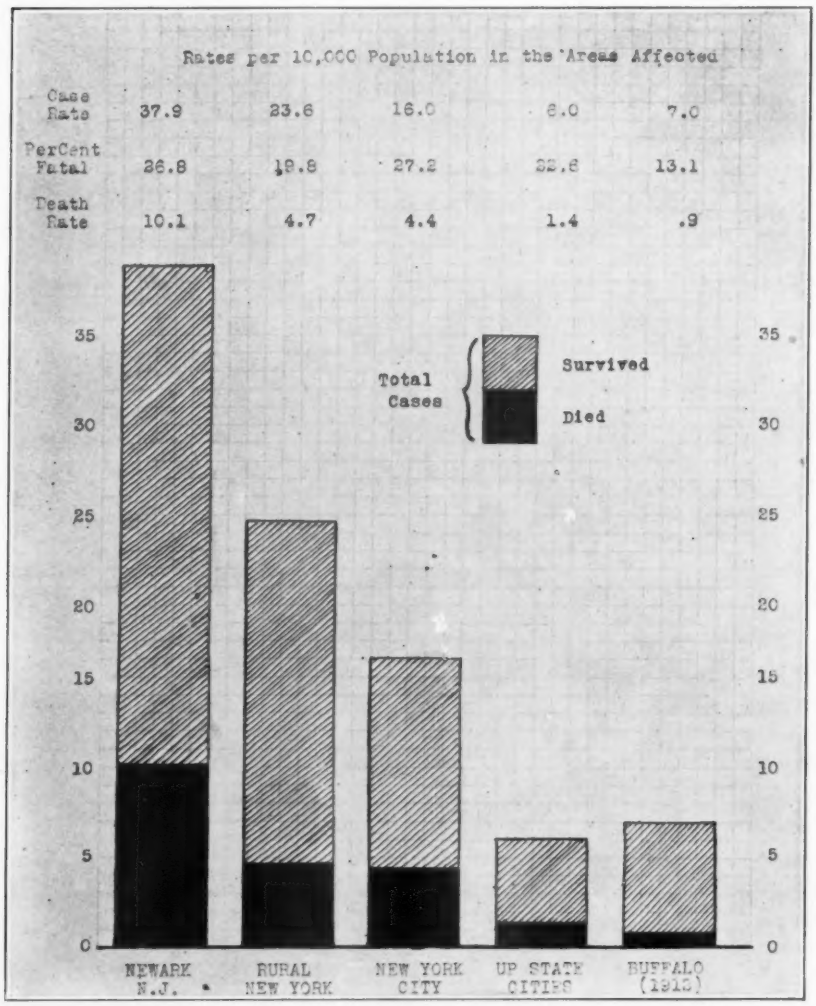

Chart 2.—Relative severity of poliomyelitis epidemic of 1916 in certain areas, judged by the comparative incidence and death rates recorded.

Whatever the explanation, apparently epidemic poliomyelitis, once it has entered a community, tends to be more widespread, but less virulent, under sparsely settled conditions of life than in the centers of population.

INFLUENCE OF SEX

The number of deaths among males was 50 per cent. higher than among females. Practically the same proportions — 60 and 40 per cent. — were observed in New York City and upstate, at the different ages, and from month to month. A study of the case figures shows that not only were boys more likely to be attacked than girls, but also that the disease is somewhat more fatal to males than to females. This is confirmed by the observed experience in the Newark epidemic.

DIFFERENCE IN AGE INCIDENCE IN CITY AND COUNTRY

One of the striking features of the epidemic was the fact that its rural incidence showed a noticeably different age distribution from the urban. An analysis of the first 7,500 cases in New York City shows that almost 80 per cent. were among children under 5 years of age; over 95 per cent. under 10, and over 98 per cent. under 16. Here, apparently, poliomyelitis was almost entirely a disease of children.

In the upstate cities, less than two-thirds of the patients were under 5 years, 86 per cent. under 10 and over 7 per cent. beyond the age of 15. In rural New York only 55 per cent. of the cases occurred among children under 5 years of age, half as many between the ages of 5 and 10, and 10 per cent. of all cases among persons older than 15 (Table 3).

TABLE 3.—AGE DISTRIBUTION OF CASES AND DEATHS FROM POLIOMYELITIS DURING THE EPIDEMIC OF 1916 IN DIFFERENT SECTIONS OF NEW YORK STATE

Age	New York City (June-Aug., 1916)		Upstate Cities (June-Dec., 1916)		Rural New York (June-Dec., 1916)	
	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.
Total cases	7,496	100.0	1,300*	100.0	2,886*	100.0
Under 5 years	5,902	78.7	840	64.6	1,575	54.6
Under 10 years	7,157	95.5	1,114	85.7	2,365	82.0
Under 15 years (N. Y. C. under 16)	7,367	98.3	1,204	92.6	2,596	90.0
Over 15 years (N. Y. C. over 16)	129	1.7	96	7.4	290	10.0
	(Jan.-Dec., 1916)					
Total deaths	2,448	100.0	294	100.0	572†	100.0
Under 5 years	1,928	78.8	174	59.2	260	45.5
Under 10 years	2,315	94.6	242	82.3	406	71.1
Under 15 years	2,375	97.0	264	89.8	462	80.9
Over 15 years	73	3.0	30	10.2	109	19.1

* Ninety-seven cases of unspecified age in the upstate cities and 182 in rural New York were distributed in proportion as the known cases.
† Includes one death of unknown age.

Due to the higher fatality of poliomyelitis among adults, when the age distribution of the deaths is considered, the contrast between the different sections is even more pronounced. It will be seen from Chart 3 that while in New York City nearly four out of every five deaths were in children under 5 years of age, and 97 per cent. of all deaths occurred among persons under 15, in the rural sections only 45.5 per cent. were under 5 years, and 80.9 per cent. under 15, with fully 19.1 per cent., or one out of every five deaths, occurring among adults, persons beyond the age of 15.

In New York City the proportion of adults in each age period remained fairly constant from month to month during the course of the epidemic, while in the state outside New York City the age inci-

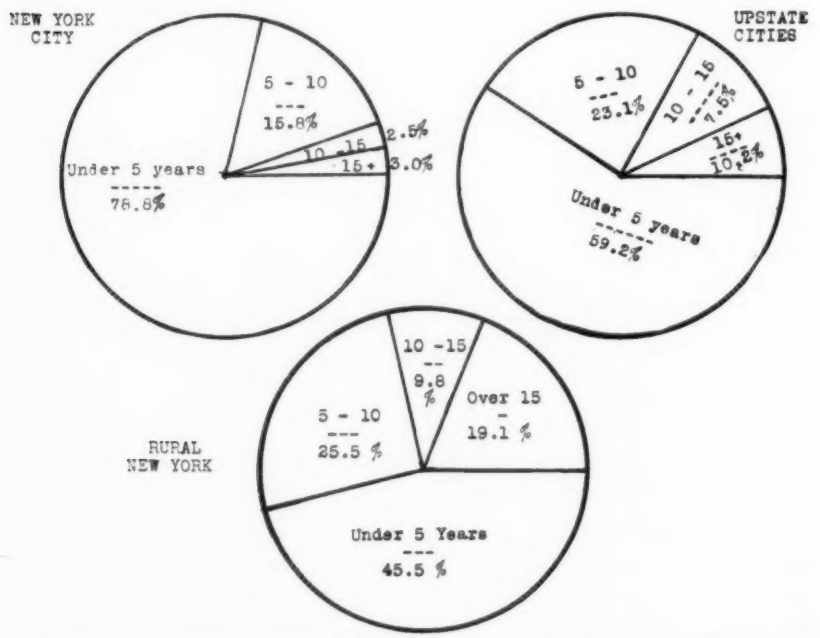

Chart 3.—Comparative age distribution of deaths from poliomyelitis during the epidemic of 1916 in New York City, upstate cities, and rural New York.

dence underwent considerable change with the progress of the epidemic. With the extension of the age limit upstate, it was observed that the proportion of deaths in the early ages decreased, and that at the later ages increased.

HAS THE RURAL POPULATION A LESSER DEGREE OF ACQUIRED IMMUNITY?

The experience of New York State with regard to the difference between urban and rural sections in age incidence of the disease is confirmed by the experience of other states during the epidemic of

1916. At the Cincinnati conference, Surgeon Frost of the U. S. Public Health Service, declared that "in rural epidemics, especially those in Iowa and Minnesota, a much larger proportion of the cases has been in the higher age groups than has been the case in epidemics occurring in large cities." Dr. Frost suggests an explanation for this difference when he says: "Certain other infectious diseases, notably measles, are largely limited to children, not because they are essentially children's diseases, but because the adult population has been more or less immunized." It seems not unlikely, therefore, that the limitation of poliomyelitis in urban epidemics almost entirely to children may be due to the fact that adults had developed a certain degree of immunity, through mild and perhaps unrecognized attacks in their early years; while the persons in more sparsely settled areas, who had been less exposed to the contagion of the disease in their childhood, had not acquired the degree of immunity which would render them able, as adults, to resist the infection when present in epidemic form.

RELATIVE FATALITY OF POLIOMYELITIS AT DIFFERENT AGES

A detailed analysis of the records of the 1916 epidemic brings out several interesting deductions as to the prognosis of poliomyelitis at different ages and under varying conditions. Chart 4 compares the proportion of cases proving fatal at each age period under urban and rural conditions. It will be seen at a glance that the disease was most fatal among infants under 1 year of age and to persons over 15. Thus, the chances of an infant surviving an attack of poliomyelitis in its first year of life were only half as good as that of a child a year or two older. After the third year the fatality rate rose with age, until among persons over 15, on the average one case out of three ended in death.

It will be noticed, too, that, up to the age of ten, the country child had a much better chance of surviving an attack than did the city child. With adults, however — that is, beyond 15 years of age — the advantage was on the side of the city bred.

THE FACTOR OF CONTACT

From an intensive study of individual outbreaks the State Department of Health has been able to obtain fairly conclusive evidence that contact is the immediate factor in the spread of an epidemic. Of 756 upstate cases that were tabulated and studied in detail, 91 cases, or 12 per cent. were definitely stated as associated with other cases in the same family, and 63 more were reported as having been in contact with other cases, making a total of 154, or 20.4 per cent. of all cases, which were known to have been associated or in contact with other or previous cases of poliomyelitis. In outbreaks of diseases which are

known with certainty to be transmitted by contact, seldom has so large a proportion of cases been definitely traced to contact.

An interesting example of a group of such definitely traced contact

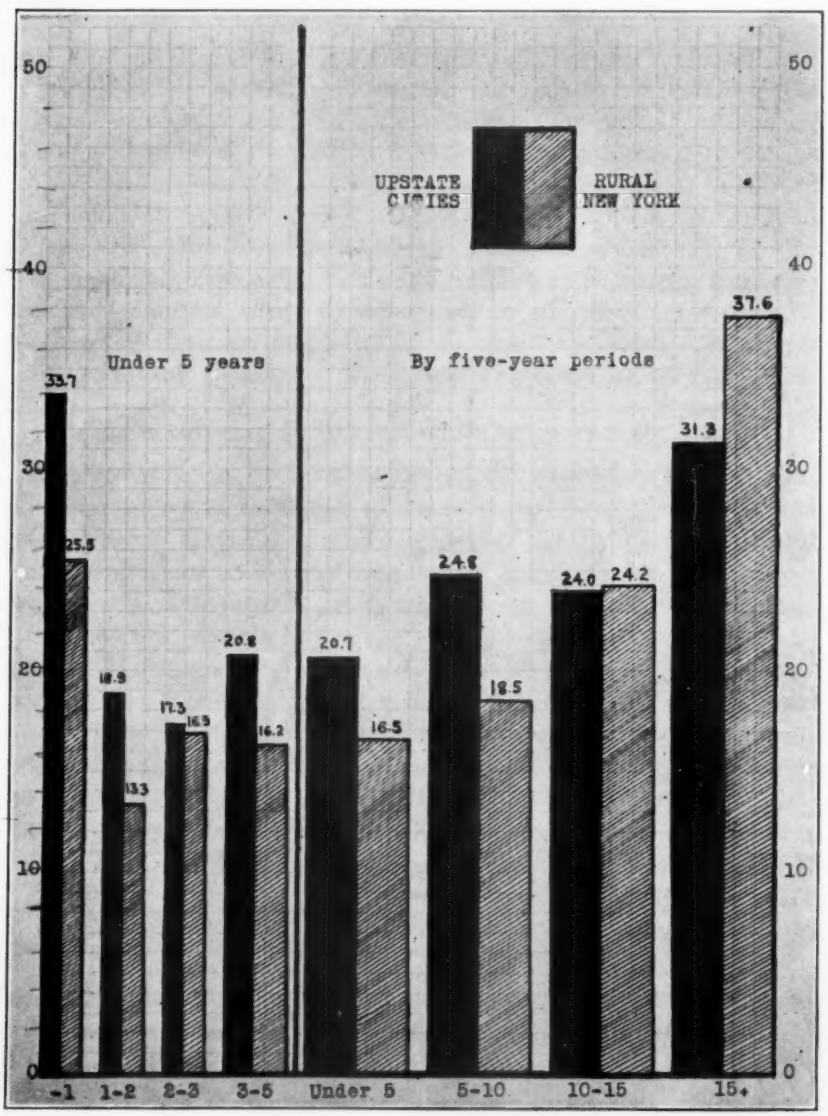

Chart 4.—Proportion of cases of poliomyelitis proving fatal at each age period during the epidemic of 1916 in upstate cities and rural New York.

cases, reported from a rural section of a northern county, is shown on the diagram (Chart 5). Note the various modes of contact by which

the disease was transmitted from C. C., the original patient of the group, to five others, of whom three died, and from them to three others closely associated with them. Note also the range of the ages — from 3 to 53 — with three of the eight known cases among persons beyond the age of 15. Of interest, too, is the period of incubation of the secondary cases. Judging from the difference between the time of exposure and date of onset, the incubation period of this group varied from eight to eleven days; half of them developing in eight days.

THE SAUGERTIES EPIDEMIC AS A TYPICAL OUTBREAK

The accompanying diagram (Chart 6), showing the incidence and association of poliomyelitis cases in the Saugerties epidemic of 1916, tells in graphic form the story of a fairly typical outbreak and how it was apparently controlled.

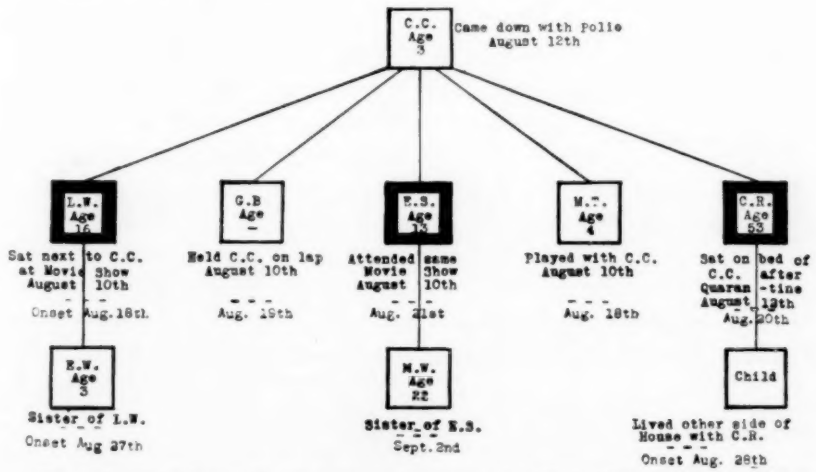

Chart 5.—Development of a contact group of poliomyelitis cases in Jefferson County during August, 1916.

Saugerties town and village form a consolidated health district, with an all-year population of 9,000 to 10,000, on the western bank of the Hudson River about 100 miles above New York City. The local epidemic seemed clearly an imported one, starting with a case coming from Brooklyn and reported to the health officer July 21. Within eight days five cases of the disease were reported in Saugerties, and then for a period of two months there developed an average of two cases a week. During the latter part of September there was a flare-up, eleven new cases developing between the 23d and 30th of the month, and fifteen cases more during the first two weeks in October.

According to the report of Dr. Charles W. Berry, sanitary supervisor, "a rigid quarantine was instituted as each case was reported,

but it did not seem very effectual in checking the disease. An investigation disclosed the fact that few of the cases were reported until actual paralysis had set in. All patients in the prodromal stage and during the period of acute symptoms before the onset of demonstrable paralysis were in full contact with other members of the household and such other persons as cared to go in and out of the sick room."

At this point, October 15, occurred the striking feature of this outbreak. A meeting of all local physicians was called that evening, and

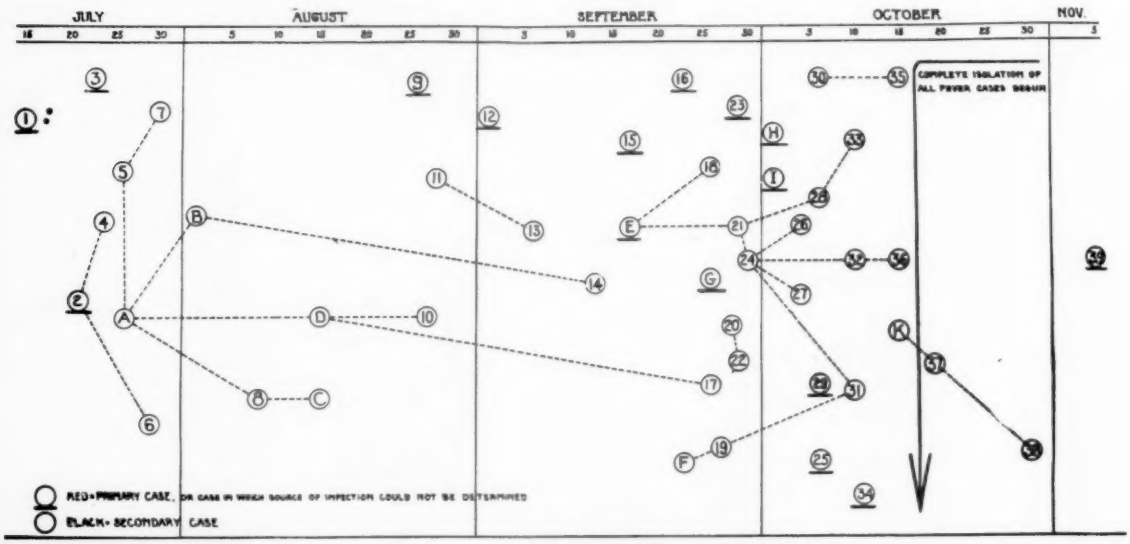
JULY
AUGUST
SEPTEMBER
OCTOBER
NOV.
COMPLETE ISOLATION OF ALL FEVER CASES BEGUN
RED = PRIMARY CASE, OR CASE IN WHICH SOURCE OF INFECTION COULD NOT BE DETERMINED
BLACK = SECONDARY CASE
PREPARED BY DR. C. W. BEARY

Chart 6.—Incidence and association of poliomyelitis cases in the Saugerties epidemic of 1916, showing the effect of complete early isolation of all fever cases after October 15.

a pledge obtained from each physician to isolate every case of fever that came under his care until a positive diagnosis of the cause could be made. This measure, coupled with the employment of a public health nurse for three weeks, who made daily inspection of all school children, followed up absentees, traced up contacts, and enforced strict

quarantine, appears to have been the means of securing effectual control of the epidemic.

Within the five days previous to the holding of the meeting on the evening of October 15, seven new cases had developed, and the adoption that night of the measures of caution described above, and a policy of complete early isolation of all fever cases, apparently had the effect of preventing further contacts with the newly developing cases, and thus halting the flare-up. After that date, only three new cases developed, two of them direct contacts already under supervision, and the third an isolated case (Chart 6).

The other noteworthy feature of the Saugerties epidemic observable from the chart is the high proportion of cases definitely traced to contact with previous cases. Twenty-six cases were traced to six foci of infection, and in only sixteen out of the total of forty-nine cases — or one out of three — could no history of contact or source of the disease be obtained.

In other respects the Saugerties instance presented the fairly typical features of an outbreak of epidemic poliomyelitis, as regards:

(*a*) Attack rate — 5 per thousand population.

(*b*) Proportion of fatalities — 16 per cent.

(*c*) Age incidence — all known cases 10 years or younger.

(*d*) Duration of acute symptoms — in twenty-three cases, 3 to 7 days (of these, ten lasted but three days).

(*e*) Incubation period — in nine out of thirteen cases with complete histories, ten days or less; in no case over two weeks.

(*f*) Proportion of cases paralyzed — nearly half of reported cases (twenty-two out of forty-nine).

PERIOD OF INCUBATION AND INFECTIVITY

From the study of a large number of cases in which a single exposure could be determined, it may be stated that the incubation period of the disease varies from four to fourteen days, with an average of about a week.

The persons taken with the disease were found to be sources of active infection for a period of at least eight days after the onset. In a number of cases infection took place as short a period as two days before the frank onset of poliomyelitis symptoms. There has been little evidence, however, of the disease being contracted from a person who had been ill longer than two weeks, which suggests the limit of the necessary period of isolation to be required for suspected cases of poliomyelitis in future outbreaks.

INFANT WELFARE WORK IN WAR TIME*

GRACE L. MEIGS, M.D.
WASHINGTON, D. C.

At this time everyone is scrutinizing eagerly the experience of the foreign countries which have been at war for almost three years. In every branch of activity we are anxious that the experience of those countries shall be of service to us, in order that we may avoid the mistakes that have been made and pursue the lines of work that have proved to be successful.

In looking over the mass of material offered on such a subject as the question of what has been done in foreign countries for the protection of infancy and maternity since the war began, it is immediately evident that a great deal of the detail of the experience recorded is worthless to us, simply because the associated conditions in the foreign countries are very different from those in our country. In each country many factors in the past have influenced the development of measures for the protection of infancy and maternity; among them are varying industrial conditions and standards of living, different methods of organization of public health and social protection, different conditions as to high infant mortality rates and falling birth rates. The countries overtaken by the war in August, 1914, and since that time, had already traveled different lengths along the road toward complete protection of infancy and maternity, and in each country different methods had been developed. It may be said at once that it would be dangerous to accept without question a great deal of the foreign experience as applicable in this country.

The greatest value to us, in a study of such foreign material is to discover certain general tendencies for good which we can imitate, and certain general difficulties which we should avoid if we can.

I should like to sum up at once what I believe these general lessons are with regard to the protection of mothers and babies in war time, as shown by a brief study of foreign reports.

In every country, and most of all in those countries which are hardest pressed — such as France and Belgium — work for the protection of infancy and maternity has been greatly increased since the war began. The decrease in the birth rates, and the fear of an increase in infant mortality rates have contributed to this interest. Especially,

* Submitted for publication June 28, 1917.
* Read before the Section on Health, National Conference of Charities and Correction, Pittsburgh, June 9, 1917.

concern for the protection of maternity as being an indispensable part of any plan for the protection of infancy, has received a remarkable impetus since war broke out. This is perhaps the one most striking feature of the foreign reports.

Another striking point is the great and successful effort which has been made in most of the foreign countries to strengthen the preventive rather than the palliative side of infant welfare work; in other words, to insure good and intelligent care of the baby by a healthy mother in her own home, rather than to give care for infants in day nurseries or other institutions. This is a notable change, certainly, from the measures employed in the wars of other days. It is the fruit of the intelligent movement for the prevention of infant and maternal mortality carried on during the previous decades.

In most countries the government itself has taken an unwonted part in such protection of mothers and babies. The line of development has not usually been toward anything new, but has been toward the expansion of work and of methods whose value for that country had already been demonstrated.

On the other hand, certain difficulties are manifest also in every country, but most of all in those countries farthest removed from the war: First, the diversion of interest and of private support from the protection of infancy and maternity to work which has a more dramatic or urgent appeal; and second, the difficulty encountered in continuing and enlarging work for infant and maternal welfare when a large part of the medical and nursing professions are called into active military service.

The chief value to this country of the foreign reports lies in this evidence they give of the added interest in the welfare of mothers and babies felt in those countries hardest pressed by the war; and of the difficulties which have been met in carrying out measures for child welfare in the face of war conditions.

The question of the increased need for the protection of mothers and babies in war time is at present felt by many people in this country to be an academic one; among the host of more dramatic appeals put forth, each one of which is claimed to be the most important in the present crisis, this question is lost sight of. You know, perhaps, the story of a meeting of women called in a large city to consider war work for women. It was addressed by the Director of the Division of Child Hygiene of the City Department of Health, who spoke of the greater necessity for infant welfare work in war time. Her speech was greeted with perfunctory applause; immediately women began popping up all over the audience saying: "Madam President! Madam President! Was this meeting called to discuss the feeding of children

or preparations for war?" "I want to nurse wounded soldiers." "But what are we going to do for our country?"

This is an extremely natural tendency; but it is a dangerous one. If the experience of foreign countries is to teach us anything, it should teach us that it would be wise to realize the importance of protecting the children, even though an extremity of need does not at present force this realization; that early in the war is the time to preserve the integrity of our work for infant and maternal welfare, before that work is disorganized by the loss of physicians and nurses. As the war goes on, doubtless many especial problems will arise; if the already established work is disorganized these problems cannot be met.

And now I shall summarize briefly the experience of a number of the belligerent countries along these three lines:

1. The influence of the war on the public protection of mothers and babies through public health nursing and infant welfare stations; through prenatal care and proper obstetrical care.

2. The influence of the war on measures to insure financial aid during pregnancy and at confinement, thus making proper care possible.

3. The influence of the war on the protection of pregnant women and nursing mothers in industry.

While certain of the measures to be described are necessary or appropriate only in the country which has developed them, certain other measures have been proved successful in all countries alike. The first of these is the establishment of centers which we may call in general "infant welfare stations," though they bear different names in the various countries. At such centers women receive advice and direction in keeping their babies well. At many of them prenatal work is also carried on; prospective mothers are given instruction and advice in prenatal hygiene, as well as medical supervision and care. The second measure, whose worth has been proved in all countries, is the teaching of the care of the mother and the baby in the mother's own home by a woman especially trained to give this teaching. In the different countries she is called an "infant welfare or public health nurse," a "sister" or a "health visitor." The rapid development of such work for the welfare of mothers and babies in foreign countries in the decades before the war has been described by Phelps[1] and Holt.[2]

ENGLAND AND WALES

Among all the countries, the experience of England since the war began is especially interesting and valuable to this country.

1. Phelps, E. B.: The World-Wide Effort to Diminish Infant Mortality, Tr. Fifteenth Internat. Cong. on Hyg. and Demog., 1913, **6**, 132.

2. Holt, L. E.: Transactions of the American Association for Study and Prevention of Infant Mortality, 1913, **4**, 24.

In England the same urgency has not been lent to the question of protecting babies as that felt in France on account of the low birth rate. And yet, in England, practically from the first day of the war, extraordinary measures have been taken to maintain and increase all means looking to the protection of mothers and babies. The part played by the national government is perhaps the most salient point in this work.

It happened that just before the war parliament was considering a grant to aid local sanitary authorities and voluntary agencies in carrying out such plans for maternal and child welfare as were approved by the Local Government Board. The grants made yearly to such work might amount to one-half of its total expense. In a memorandum bearing the interesting date of July 30, 1914, the Local Government Board gave the details of what such schemes should include, divided into measures for antenatal, natal and postnatal care. The systematic home visiting of infants and young children was dwelt on, as well as the carrying on of centers for infant and maternal welfare. Especially emphasized also were the need of coordinating public and private work; the importance of providing proper prenatal and obstetrical care; and the desirability of giving greater attention to the care of the child between infancy and school age.

This grant before the war fortunately placed in the hands of the Local Government Board a powerful means for stimulating and helping work for infant and maternal welfare after war began; and the board has used it with great success. Throughout it has taken the stand that in war time, in spite of the general need for economy, no economy should be exercised in this direction. There is evidence that in a good many communities, on account of lack of money and private support, the authorities or voluntary agencies have been slow to increase their work, or to undertake new work. These difficulties the Local Government Board has largely overcome. It has gone on with the greatest determination towards its acknowledged goal — to have systematic supervision through the work of health visitors for all babies born who need care. It was estimated that to accomplish this, one full-time health visitor for every 500 births reported annually was necessary.[3] The passage, in 1915, of the Notification of Births Extension Act was a powerful help toward this end. Through this act the immediate notification of all births to the health authorities was made compulsory in all parts of the British Isles; this notification within thirty-six hours makes possible the immediate visiting of the mothers of all babies born who need care.

The Local Government Board has succeeded so well that in the reports of the board and of its medical officer, Sir Arthur Newsholme,

3. Rep. Grt. Brit. Local Govt. Bd.: Maternity and Child Welfare, 1916, p. 16.

for 1915-1916, the statement[4] could be made that health visitors were now employed or subsidized by all the metropolitan boroughs except Camberwell, and by all the county boroughs except Gateshead. While in March, 1914,[5] there were only 600 health visitors employed by local authorities, this number had increased to 812 at the end of 1915, and to about 1,000 (including full-time and part-time workers) in 1916. This represented one health visitor, approximately, to every 800 births annually.

The increase in work for the protection of maternity through the careful supervision of midwifery and the provision of free prenatal care and skilled confinement care for those unable to pay for it, is less easily shown by figures; but this increase is very manifest in the reports.

In addition, the extension of infant welfare work to all children up to school age has been a successful part of the campaign.

Summing up the work of the Local Government Board, Sir Arthur Newsholme makes this statement in his report for 1915-1916:[6]

> The war has had the effect of directing greatly increased attention to means for improving the health of mothers and their children during the first five years of life. During 1915 work with this object has been much increased, though some local authorities still remain inert, and appear to be unwilling to realize that the truest national economy can only be secured by saving life and improving health by all practicable means.

That the Local Government Board is planning still greater efforts is shown by recent press articles and by correspondence. Lord Rhondda, soon after his appointment as president of the board, declared it as his belief that the lives of 1,000 babies[7] could be saved each week. And now we hear that the board is taking an active part in carrying out a National Baby Week in July, 1917. There is a National Baby Week Council, of which the Prime Minister is president, and the president of the Local Government Board, chairman. The board is sending suggestions as to the campaign to mayors, medical officers of health and town clerks in 250 districts. It is estimated that 800 to 1,000 different campaigns will be held. The National Baby Week Campaign, like that in this country last year, is to be one of education.

Another branch of the government has also taken part in infant welfare work since the war began. This is the Board of Education,

4. Rep. Grt. Brit. Local Govt. Bd.: Forty-Fifth Annual Report for 1915-1916, Part 3, p. 13.
5. Grt. Brit. Local Govt. Bd.: Supp. containing Report of Medical Officer for 1915-1916, p. 34.
6. Grt. Brit. Local Govt. Bd.: Supp. containing Report of Medical Officer for 1915-1916, p. 4.
7. Editorial: The Times, London, April 2, 1917.

which offers grants in aid to schools for mothers and to day nurseries, and has set up standards for the work of the institutions receiving this aid.[8]

Such work as above described is educational; it is directed against one of those two primary causes of infant mortality, ignorance and poverty.

The forces affecting the economic conditions of the mothers and their babies are of course of primary importance. Conditions which enable a mother to provide herself with proper care during pregnancy and confinement, and which make it possible for her to nurse her baby and care for him in her own home are the most powerful agencies of all for maternal and infant welfare. We may say that just compensation to dependents of soldiers is, after all, the greatest war measure that can be taken for the preservation of infant life in the presence of war.

I shall not here discuss the relation of economic conditions in England since the war began to the welfare of mothers and babies. I do wish, however, to say a word on one phase of this question: that is, the maternity benefits under the National Insurance Act. Like most of the other countries in which some system of maternity insurance was in force at the outbreak of the war, Great Britain has continued this work during the war, in the face of some difficulty. In 1914 the health insurance fund was facing a deficit because of the unexpected high rate of sickness among married women; and £500,000 were appropriated by parliament to cover this deficit. In the following year £150,000 were appropriated for the same purpose.

From English reports we have been unable as yet to obtain any definite information as to the relation of the profound change brought about in industrial and economic conditions by the war to the employment of pregnant women and nursing mothers in factories, especially in munition factories. While the statement is made that many mothers of young children are being employed in factories, here, as in other countries, definite figures seem to be lacking to show whether this is actually the case.

England, like all foreign countries, is watching its infant mortality rate and birth rate with as much concern as its casualty rates. In England and Wales, as elsewhere, the birth rate has fallen markedly; the infant mortality rate, as giving evidence of the success or failure of all the measures spoken of, has been watched, therefore, with all the more eagerness; and to everyone's great satisfaction it reached in 1916 the figure of 91 per 1,000 births, the lowest for any year on

8. Grt. Brit. Bd. of Education: Annual Report of the Chief Medical Officer for 1915, p. 24.

record. This fall was not immediate, however; the rate for 1914 was about the same as that for the year previous (105 as compared with 108); that for 1915 rose slightly, being 110. The fall in 1916 has aroused enormous interest. It is attributed to many things. A comparatively cool summer no doubt played some part. To the increase in wages great importance is also ascribed. A great deal of the credit is given, and no doubt with justice, to the remarkable increase in the measures which have been taken to protect infancy and maternity.

SCOTLAND

In Scotland a good many of the same measures have been carried out as in England and Wales; and here, too, the infant mortality rate declined very markedly in 1916; 111 per 1,000 births in 1914, it rose to 126 in 1915, and then fell to 97 in 1916, which was 9 points less than the lowest record ever before recorded for Scotland.

GERMANY

Our knowledge of conditions in Germany is far less complete than of those in England. We have but little information covering the last year and a half.

I can best sum up the effect of the war on work for the protection of mothers and babies in Germany by quoting reports of Drs. Langstein and Rott, director and assistant at the Kaiserin Auguste Victoria Haus zur Bekämpfung der Saüglingssterblichkeit im Deutschen Reiche. This institution is the official headquarters of the movement for the protection of infancy in Germany. Their articles published in the early part of 1915 tell us only of conditions in the early months of the war; but they are interesting as showing the lines on which the work was laid out in Germany for the war period. Dr. Langstein[9] says:

> Certainly never was the truth of the saying that children are to be regarded as the most valuable capital of the state more clearly apparent to every one than at the beginning of the war. The greatest care of all those who were engaged in social work was the preservation of all those measures for the protection of children which Germany has established in the last few years, and which may serve as models, and to which we are primarily indebted for the fact that a decrease in the infant mortality rate has been attained.

Langstein speaks of the fact that in the early days of the war attention was directed primarily to care for the army and for the wounded. Nevertheless, very soon after the beginning of the war a meeting of representatives of infant welfare work in Berlin took place at the Reichstag. The resolution to continue infant welfare work and to

9. Langstein, L.: Die Bedeutung des Kriegszustandes für das Schicksal der Kinder. Ztschr. für Säuglingsschutz, 1915, **7**, 41.

increase it was expressed, and this resolution was communicated to other German communities. The effort has been successful. Langstein continues:

> As far as information is available, it may be said that the care of infants in Germany has shown in time of war on what a firm foundation it rests. The work, which was established in time of peace, has in general continued. The people who were in charge of the work have been conscious that it is just as important a patriotic task to serve children who need care as to serve the soldiers in the field. I have been informed, it is true, that in certain regions of East Prussia, evidently under the stress of a dread of hostile attacks which are continually recurring, the work for the protection of infants has been discontinued.

Langstein also speaks with regret of the closing of the Neumann clinic for children in Berlin.

A few months later, in June, 1915, Dr. Rott[10] reported on a questionnaire sent to officials and private organizations in over 375 communities of over 15,000 inhabitants, and to 788 infant welfare centers, to 266 institutions for the care of mothers and babies, and to 271 day nurseries. The object of this study was to find out the effect of the war on the great system of infant welfare work which had been developed in Germany before the war. Eighty-one per cent. of the communities answered the questionnaire. It was found that in only 2 per cent. of these communities had work decreased; in 10 per cent. it had increased, and in the remainder, 88 per cent., it had continued unimpaired. The centers which had been closed were those which depended on private subscription. The loss of physicians and nurses to war work was remarked on in many communities; especially characteristic of private associations was a tendency of the sisters who had been carrying on infant welfare work to leave it for active military duty. Dr. Rott described this tendency as unnecessary and deplorable. He says:

> The lack of physicians and nurses is to be regarded as a direct result of the war; the scarcity of the former may be considered, for the most part, as necessary and unavoidable; that of the nurses, however, is by no means unavoidable. It is certainly a deplorable fact that a large number of infant welfare sisters and attendants at the beginning of the war offered themselves for the care of the wounded (although in this department there was an oversupply of nurses), and so withdrew themselves from the extremely necessary infant welfare work.

One section of Dr. Langstein's report is especially significant.

> Extraordinarily interesting, and not foreseen by many was the fact that since war began less use has been made than in times of peace of asylums, especially of infants' homes, intended for the care of healthy infants, and also of day nurseries. People at first thought that this would be different; and many promi-

10. Rott: "Die Einwirkung des Krieges auf die Saüglingssterblichkeit und die Saüglingsschutzbewegung," Ztschr. für Saüglingsschutz, 1915, **7**, 177.

nent women were convinced that the need for asylums and day nurseries would be so great that the establishment of new ones was an urgent necessity. . . . An absolute need for the establishment of day nurseries and homes (*Krippen und Heimen*) did not exist, as the course of the war has taught; and I may say that this is the finest proof to what a high level the care of infancy has attained in at least a number of large cities.

Dr. Langstein goes on to say that this was not the case everywhere. In Vienna, for example, there were few institutions for the care of children; and here their rapid establishment in troubled times caused great difficulty.

The reason why certain day nurseries were little used was because the mother, now unemployed, was in the position to keep her child with her and to devote herself to its care. It seemed to many thoughtful people that to strengthen this bond, which the war has tied anew, was a task fine and important, not only for the present, but for the future.

Dr. Rott also speaks of the establishment of many day nurseries after the war began, because of the impression that women would go to work at once. Many remained unused because of the lack of employment for women; where the nurseries were used the results were bad, because of the fact that they were carried on with low standards as to space, equipment and personnel. "Happily, some of them were soon closed," he remarks. He is convinced of the benefit of offering a "stillprämium," or allowance to a mother who nurses her baby.

Both speak of the mistake which was made early in the war, when an appeal was made to rich families to undertake the support of children away from their mothers. Dr. Langstein says that, on the other hand,

It seemed to the discerning that the only way to meet the need was through strengthening the bond between mother and child, namely, by giving financial aid to the mother or to a good caretaker who desired to keep the child with her, but who was unable to do it on account of financial difficulty.

He says that this fact has been taken into account in Berlin by the special committee for the care of mothers and infants, which is part of the Red Cross. The committee stipulates that the mother shall bring her baby regularly to an infant welfare station. It is said:

This fund gives to all mothers, who are shown to be in need, and who during the course of the war wish to take care of their children in their own homes, a monthly allowance; and fulfils therewith a task which is not only successful as regards health, but is also a great social task. I hope that the work of this committee may not end with the peace which we hope will soon come.

Dr. Rott reviews other measures which have been taken which seem to him of paramount importance for the welfare of mothers and babies in Germany. First among these he speaks of the government provision made for the maintenance of families of soldiers, supplemented by the provision made by communities.

Secondly, he speaks of the *Reichswochenhilfe,* the maternity and nursing benefits now paid by the imperial government. The effect of the war on maternity insurance in Germany has been an interesting one. In August, 1914, the Reichstag, fearing the effect of the war on the solvency of the health insurance societies, curtailed greatly the maternity benefits. It soon became evident that the sickness insurance societies were less affected by the war than had been feared; also the need for the protection of wives and babies of soldiers seemed very great. Therefore the imperial government assumed the burden of maternity benefits for wives of soldiers who would not otherwise receive benefits, and these were increased over the benefits previously paid. The benefit paid for twelve weeks after confinement to mothers who nurse their babies was a special feature. Another act in 1915 still further increased the number of women to be benefited. The government is now expending 5,000,000 marks a month for this purpose.

No German reports have been available on the subject of the development of work for the protection of babies in Germany during the second year of the war. However, light is thrown on this question by the "Report on Milk Supply in Germany,"[11] prepared by Prof. A. E. Taylor of the American Embassy staff in Berlin in 1916. He finds no evidence that infants in Germany have suffered because of the decrease in the milk supply since the war began. Measures had been taken early to prevent any such hardship. It is said:

> The question of the adequacy of the supply of milk for the needs of the German empire was realized early in the war and regulations were promulgated by the authorities for the purpose of accurate control. The use of milk, apart from that applied to the making of cheese, was grouped under four headings:
>
> (*a*) Use of milk by the nursing mother and the weaned infant.
>
> (*b*) The use of milk by children from the second to the twelfth or fourteenth year.
>
> (*c*) The use of milk by the sick under the care of physicians.
>
> (*d*) The use of milk as a beverage by healthy adults.
>
> The use of milk as a beverage by healthy adults was regarded, under the conditions at present pertaining in the German Empire, as luxury; and it was ordered that no milk should be dispensed for this purpose until all requirements under the three first-named headings (*a, b, c*) had been fully supplied. The reduction in the milk supply has naturally been felt most keenly, and indeed almost entirely, in the cities, and in many cities in the empire there has been during the past winter little or no use of milk as a beverage by healthy adults.

As explaining the decline in the infant mortality rate during 1915 Professor Taylor says:

> This extremely favorable and impressive reduction in the death rate of children under 1 year was achieved through the cooperation of several agencies. It

11. Taylor, A. E.: Report on Milk Supply in Germany, U. S. Department of State, 1916.

is an axiom with workers in social service that the fewer the children the better the care which they receive; and this undoubtedly has held in Germany, particularly in view of the insistence placed by the press on the preservation of the future generation. Added to this was the fact that the summer of 1915 presented a subnormal mean of temperature, which, here as everywhere, operates to reduce the so-called intestinal diseases of infants. Lastly, the social service organizations in the cities of Germany have, during the past winter, reached a point of unequaled thoroughness and excellence. In the city of Berlin alone there are some 6,000 workers actively engaged in social service. Under these circumstances it has been possible to uncover and combat practically all instance of need in the care of infants. There is nowhere to be found any evidence of any reduction in the milk supplied to nursing mothers and infants in Germany during this period of time.

In Germany great attention has been given to the question of the effect of the war on the birth and infant mortality rates. The infant mortality rate for 1914—164 per 1,000 births—was a marked increase over the rate for 1913, which was 151 per 1,000 births. Dr. Langstein prophesied that this increase would probably be shown by the figures for 1914-1915 because of the prevalence of summer diarrhea among infants in August and September, 1914. This he ascribes to the hot summer and to the effect of the many conditions attendant on the breaking out of war.

The drop in the birth rate for 1915 showed markedly the effect of the war. The infant mortality rates, however, showed also a decrease, at least in the larger cities. Official reports for the infant mortality rates of 1915 and 1916 for the whole of Germany have not been available. The *Deutscher Reichsanzeiger*[12] reports an interesting official study which was made of the figures for births and infant deaths in the twenty-five large cities of over 200,000 inhabitants for the three months from August 1 to October 30, 1915. As compared with the same period of the year before, there was a decrease in the number of births of 15,457. The decrease in the number of deaths under one year was 6,354. The statement is made:

The decrease in the number of births was therefore almost one-half counterbalanced by the lowering of the infant mortality. . . . These relations would lead one to suppose that in general with the decrease in the number of live-born, an increase has occurred in the welfare work undertaken in certain cities; or that this work became more efficient because it reached better the infants needing help than when the number of infants was larger. It is true that the favorable weather conditions of the summer months of the year 1915 were in addition a favorable influence.

Dr. Taylor's report gives information on this point covering a later period in the second year of the war:

The *Kaiserl. Gesundheitsamt* keeps a special record of the death rate of children under 1 year. These figures are published up to January 1, and the unpublished figures up to April 1 have been kindly placed before me in the

12. Deutscher Reichsanzeiger, Feb. 8, 1916, No. 33.

Gesundheitsamt. They display, in a most striking manner, a reduction in the death rate, a reduction unparalleled in Germany in time of peace. In fact, viewing the figures as a whole, it is clear that the reduction in the death rate under the first year of life has been so large as to have compensated for half of the reduction in the birth rate during the past year.

FRANCE

In France the available reports deal chiefly with the work for maternal and infant welfare in Paris. This was organized early in August, 1914, with Paris under military government, with the title *Office central d'assistance maternelle et infantile.* The published program of this office was this:

> During the entire war, and in every part of the military government of Paris to assure to every woman who is pregnant, or who has a baby less than 3 years old, the social, legal and medical protection to which she has a right in a civilized society. To be sure that no woman is ignored and no child is forgotten.

Delegates of the office were installed in the eleven maternities of Paris of the *Assistance publique* (the official agency in charge of charities in Paris), to which most needy women come for confinement, and in the *mairies* to which they apply for medical assistance when they are confined at home. The object of this office was evidently to make the work already carried on immediately available to the women needing help; to stimulate all organizations and institutions to increase their work, and, where necessary, to establish new work.

Professor Pinard, in his reports on the protection of infancy in Paris during the first five months and during the first and second years of the war, has reviewed the measures taken. He says[13] that the medical protection of mothers before, at and after confinement, and of babies had never been so good in Paris as during the first five months of the war. The provision of free hospital beds for confinement was greatly increased; free consultation centers for pregnant women were established at every medical center. Military ambulances were provided to carry women to the hospitals. *Cantines maternelles* gave meals to pregnant women. The infant welfare stations or *gouttes de lait* continued their work. The lack of milk for babies who were bottle fed was at one time a great danger. This was obviated to some extent by the provision by the municipality of pure milk from the Paris herd especially for babies. About 12,000 liters of this milk were distributed daily. Pinard,[14] in later articles, reviews the measures which have been carried out by the government and the *Assistance*

13. Pinard, A.: Bull. de l'Acad. de méd., Paris, 1915, **73,** 220.

14. Pinard, A.: Bull. de l'Acad. de méd., Paris, 1915, **74,** 343; ibid., 1916, **76,** 540. Pinard, A.: Ann. d'hyg. pub., 1917, **27,** 76.

publique in order to insure financial support for pregnant women and the mothers of young babies. These are:

1. The military allowance given to the mother of the children of soldiers whether legitimate or illegitimate.

2. The government maternity grant, which was established by the act of June 13, 1913.

3. The grant given by the government to large families.

4. The help distributed by the *Assistance publique,* and by the maternity hospitals, whose object is to enable the mother to care for her baby herself.

The degree to which the public provision for confinement care has been used seems the most astonishing fact in the figures given by Pinard; they reveal a need for economic assistance on the part of almost all the mothers confined, which creates a profound impression. While in 1913-1914, 78.5 per cent. of all the births in Paris were cared for either by the maternity hospitals of the *Assistance publique,* or through its medical service outside the hospitals, this figure rose in 1915-1916 to the astonishing percentage of 95.2 per cent. That is, in 1913-1914, 10,465 women out of 48,917 went through confinement in Paris without the help of the *Assistance publique;* in 1915-1916 only 1,250 out of 26,179. The figures show, too, the fall in the birth rate in Paris; in 1915-1916 the number of births was only a little more than one half what it was two years before, in 1913-1914 (48,917 in 1913-1914; 26,179 in 1915-1916).

The results of this work in Paris, Pinard sums up in his papers. The results for the first year from Aug. 1, 1914, to Aug. 1, 1915, were very encouraging. The infant mortality rate among the babies remaining in Paris declined; the maternal mortality rate fell as did the still-birth rate. The death rate under 2 years, however, remained about the same. There was a remarkable decrease in the number of babies put out to board outside of Paris. In 1913-1914, before the war, the number of children who did not have the benefit of their mother's care for this reason was 14,925; in 1914-1915 it was only 4,954. The percentage of new-born babies abandoned was considerably lower in the first year of the war than previously.

Dr. Pinard's report for the work during the second year of the war, Aug. 1, 1915, to Aug. 1, 1916, shows results no longer so encouraging. While the work described above was continued or was increased (the number of women helped at confinement now being 95.2 per cent. as has been said), the work was less successful in some respects, although the infant mortality rate among the babies remaining in Paris was still less in the second year of the war than in the first. There was, moreover, a decline in the mortality in the early months of life and from

diarrheal disease. The last named decrease Pinard attributes to the work done: (*a*) to protect babies before birth; (*b*) to insure in a larger number of cases that the mother should stay with her baby and nurse him; (*c*) to provide good milk when breast feeding was impossible.

On the other hand, the death rate of children under 2 years was higher than before the war. The rates from measles and whooping cough increased. Most disquieting to Pinard are the following facts as shown by the statistics of Paris for the second year of war. The percentage of new-born infants abandoned increased greatly, so that it was actually higher than before the war; also the number of babies put to board outside of Paris was much greater than in the first year, though still less than before the war. The stillbirth rate and the maternal mortality rate had also increased.

Pinard draws from these results conclusions which are destined to make his report a famous one, for they have given rise to a heated and protracted controversy. Pinard believes that the unfavorable results obtained in Paris during the second year of the war are only to be explained by the entrance of pregnant women and nursing mothers into industry in factories, especially munition factories. Pinard, therefore, at the meeting of the French Academy of Medicine, at which he had read his report, proposed that resolutions should be passed by the Academy advising the government to take the following measures: To forbid that any woman in France who is pregnant, nursing her baby or confined within six months should be employed in factory work; and that every French woman pregnant or nursing a baby of less than a year should receive on her demand, an allowance daily of 5 francs.

The discussion that arose in the Academy of Medicine over this speech of Pinard and the report of a committee appointed to consider it was continued at many of the weekly sessions from Dec. 5, 1916, until March 13, 1917.

It is evident that both sides in this controversy had equally at heart the same object: the protection of infancy and maternity in France; they differ greatly as to the method to be employed. One side favors abolition of factory work, especially in munition factories, for pregnant women and nursing mothers; the other side favors its regulation. One point seems clear on reading the reports of this discussion: there is little actual knowledge of whether or not the number of pregnant women and nursing mothers now employed in factories, especially munition factories, is actually as great as the conclusions of Pinard and his followers would lead one to suppose. The only evidence that is available tends to show that this number has been overestimated. M. Bonnaire reports that at the Maternité in one month, November 10 to December 10, 1916, 7 per cent. of the 445 women confined had been

working in munition factories. Most of these had, however, obtained a change of work or had stopped work because of their condition. One speaker, M. Bar, in the discussion at the Academy related the results of an informal investigation in a few big munition factories as to the number of women employed who were pregnant or had children under one year. He felt sure that evidence as to the number of pregnant women was undoubtedly incomplete. He concluded, however, that not more than 1 per cent. of the women working in these factories was pregnant, and not more than 2 to 3 per cent. of the women had children under 1 year.

During the discussion, the resolutions passed in December, 1916, by the committee on female labor appointed by the Ministry of Munitions regarding the protection of pregnant women in munition factories were discussed; these deal with the forbidding of night and overtime work, work in the last four weeks, and certain forms of labor to pregnant women; also with medical supervision of such women.

After prolonged debate, the resolutions of Pinard were defeated, and resolutions were passed by the Academy based on the report of the committee appointed. These resolutions embody still further recommendations as to the regulation of the work of pregnant women and nursing mothers in munition factories, and in order to favor maternal nursing advise the establishment in factories and munition factories of rooms where working mothers can nurse their babies.

The latter institutions were especially discussed during the debate. Pinard said that while he had sometimes favored their establishment, that he considered them very difficult to manage well. He knew of only one which was satisfactorily equipped and conducted, and that was carried out at a high cost, estimated at about 5 francs a day per child. Lesage, in numerous publications, has urged the establishment of these rooms in munition factories, and has emphasized the great precautions which must be taken in carrying them on.

BELGIUM

About the work for maternal and infant welfare in Belgium, Dr. Lucas'[15] report gives us immensely interesting information.

Information as to death rates is of course difficult to secure in Belgium, but all the figures available point to an actual decrease in the infant mortality rate in at least the large cities of Belgium (with the exception of Mons) since the beginning of the war.

Dr. Lucas writes:

> General solicitude for the children has resulted in an actual improvement in infant conditions to a point above the normal. . . . It is generally evident that

15. Lucas, W. P.: General Health Conditions in Belgium After Two Years of Relief Work, Jour. Am. Med. Assn., 1917, **68**, 27.

infant conditions are on the whole better than normal, that class having been the object of great solicitude since the beginning of the war. A great many institutions have been created during the war to care for the children. The organization of the work for the children has been divided into canteens:

1. Canteens for nursing and expectant mothers. . . . There are over 13,000 nursing and expectant mothers receiving these dinners.

2. Canteens, often in conjunction with maternal canteens, have been instituted for infants under 3 years, in which there are over 52,000 infants receiving milk and cereals.

Both these types of canteens have undoubtedly had a great influence on the reduction of infant mortality in Belgium. There is no question that today, in Belgium, more is being done for the mother and the child than was ever done before. Previous to the war there were only two maternal canteens in the whole of Belgium; today there are over 329 canteens for infants. These canteens, in connection with the educational work, the medical supervision which all the canteens have, and the careful regulation of the dietary, both in the canteens and by an extensive system of visiting nurses, in the homes, have undoubtedly had a marked effect on this great reduction in infant mortality.

The fact that the educational and preventive work of these canteens has been made so marked a feature in the face of conditions such as those present in Belgium is, I believe, one of the greatest triumphs of preventive infant welfare work that can be thought of.

There is not space to speak here of work in Italy and Russia; moreover, our information about these countries is meager.

The experience of such countries as New Zealand and Canada is interesting to us in many ways, especially because their situation, far removed from the actual scene of the war, resembles that of this country. In these countries, from the small amount of evidence at hand, it would seem that there has been no such increase in the care for the protection of maternity and infancy as that which has occurred in the countries already spoken of. On the other hand, there is much evidence that the work has suffered greatly from the loss of physicians and nurses called to active military duty.

In the report published in 1915 of the New Zealand Society for the Health of Women and Children,[16] which has done such remarkable work, we read:

While each individual member of the society and each Plunket nurse has been working more strenuously, the society has temporarily limited the sphere of its operations as regards the establishment of new residential centers for Plunket nurses. In some districts where sufficient funds had been collected to enable the local committees to apply for the government grant of 24 shillings for each pound subscribed, the Central Council asked them to hold the matter over in the meantime, and to work quietly without a Plunket nurse, getting an occasional visit from the nearest nurse as a stimulus to continued effort. This was done at the request of the government, in view of the greatly increased public expenditure which the war was involving.

16. The Society for the Health of Women and Children, Eighth Annual Report, 1915, p. 3.

And in the report[17] published in 1916 it is said:

> During the year a great many of our nurses who volunteered for war service left for the front, and several of the branches have been obliged to avail themselves temporarily of the services of Karitane nurses while new Plunket nurses were being trained. As arranged with the government authorities when war broke out, no new residential centers for Plunket nurses have been created during the year.

From Canada the reports of the Victorian Order of Nurses gives us an indication of the effect of the war on the work of this society which carries on infant welfare and prenatal nursing in connection with general public health nursing, not only in the large cities, but in the most isolated rural districts of the vast stretches of western Canada.

In her report for 1915[18] the chief superintendent, Miss Mackenzie, says:

> The war has affected the order in several ways—our increases are not as great, and we have not opened up new districts in cities, many of which were ready to organize when the war broke out. In many of these districts the order is being used and appreciated more than ever before, and doubtless that will be still more noticeable as time goes on.

During 1915 twenty-eight of the band of 292 nurses of the order doing active work resigned to go overseas for war duty.

The 1916 report describes especially the effect of the war on the movement for public health nursing, especially maternity and infant welfare nursing in the isolated western country districts. This movement received a great impetus in 1912 through the establishment of the Duchess of Connaught's fund for nursing in new and sparsely settled districts. The report says:[19]

> A promising beginning was made, but since the war came on us, it has become harder and harder to secure nurses for these outposts, and many districts ready to receive nurses have had to go without them.

Miss Mackenzie writes in general, however, that conditions are now better than the year before. She speaks of the shortage of nurses as one of the most serious handicaps, but says that this is gradually improving. In spite of this shortage she says: "The most gratifying development in the Victorian Order during 1916 is to be found in the country scheme." She writes also:

> When the war broke out many of our committees were more or less panicky, fearing that it would be necessary to suspend V. N. O. activities until after the war, and town districts where branches were to have been organized

17. The Royal New Zealand Society for the Health of Women and Children, Annual Report, 1916, p. 6.

18. Victorian Order of Nurses for Canada, Report of the Board of Governors for 1915, p. 13.

19. Victorian Order of Nurses for Canada, Report of the Board of Governors for 1916, p. 14.

dropped all plans for the time being. Now, the past year has shown a wonderful change; the panicky condition is not noticeable at all, and the reports, with very few exceptions, show increased visits, increased fees and contributions.

She speaks of an increase in the work for infant and prenatal nursing.

Canada has given evidence of its interest in infant welfare through its Baby Weeks. The Children's Bureau last year had reports of three successful celebrations in Canada; and this year has heard of plans in seventeen cities, including large cities such as Montreal, Hamilton and Calgary, as well as small settlements in the far west.

Canada has shown a far-sighted liberality in its provisions for soldiers' families. These are described in Mr. Paul Kellogg's articles in the *Survey*,[20] and in a report by Mr. Wolfe recently published by the Children's Bureau.[21]

CONCLUSIONS

To me, as I have said, this brief study of how war has effected the protection of maternity and infancy in many countries seems to give us in this country some practical suggestions now that the United States is also involved in the war. These are:

1. No hasty conclusions should be drawn that the war makes immediately indispensable in this country such palliative measures as the increase of day nurseries or the supervision of pregnant women working in factories, to which dire necessity has driven certain foreign countries. Study is necessary to show how present and future economic and industrial conditions will affect the number of pregnant women and of mothers of young children employed in factories; and what measures are needed under these conditions.

2. The chief preventive measure for protecting babies is to insure their intelligent care and nursing by healthy mothers in their own homes.

3. The disorganization of infant welfare work through the loss of physicians and nurses especially trained for it, is an imminent danger, and should be avoided if it can be done. In view of the greater demand for nurses, every effort should be made to enlist a large number of candidates for hospital training courses.

4. The preventive work for infant and maternal welfare, already established, should be strengthened and extended; and nothing should be considered more important in war time.

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20. Kellogg, P.: A Canadian City in War Time, The Survey, 1917, **37**, 677, 709 and 739.

21. Wolfe, S. H.: Care of Dependents of Enlisted Men in Canada, U. S. Children's Bureau, Pub. 25.

THE URIC ACID CONTENT OF THE BLOOD IN THE NEW-BORN *

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It has long been known that the uric acid excretion in the urine of children during the first few days of life is both relatively and absolutely high. Reusing[1] determined the daily excretion of this substance during the first seven days of life of six normal children and found that it reached its maximum of 0.083 gm. on the third day. He used the analytical method of Gowland Hopkins.[2] Schloss and Crawford,[3] using the procedure of Folin and Shaffer,[4] determined the uric acid excretion of nine infants each day up to and including the ninth day, and found in four of the cases that the excretion reached its maximum on the third day; in two cases on the second day; in two cases on the first day, and in one case on the fourth day. Their results are confirmatory of Reusing's. Schloss and Crawford also pointed out that the urinary excretion of phosphorus was highest during the first three days of life and believed that this indicated a common source of origin of phosphoric acid and uric acid, the nucleoproteins. They did not, however, determine the fecal phosphoric acid excretion. The purin content of the colostrum they found to be too slight to account for the high uric acid excretion.

It is well known that during this period when the uric acid excretion is highest, rapid morphologic changes occur in the blood of the new-born. The disappearance of the nuclei of many of the red cells, the change in proportion from a predominating number of polymorpho-

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* A preliminary report of this work was made at the annual meeting of the Federation of American Societies for Experimental Biology, December, 1916, in New York City.

* Read at the meeting of the American Pediatric Society, White Sulphur Springs, W. Va., May 28, 1917.

1. Reusing, H.: Ztschr. f. Geburtsh., 1895, **33,** 36.
2. Hopkins, F. G.: Guy's Hosp. Rep., 1891, p. 299.
3. Schloss, O. M., and Crawford, J. L.: AM. JOUR. DIS. CHILD., 1911, **1,** 203.
4. Folin, O., and Shaffer, P. A.: Ztschr. f. physiol. Chem., 1901, **32,** 552.

nuclear neutrophilic cells to a corresponding predominance of lymphocytes (Carstanjen[5]), and the striking decrease in the leukocyte count of the peripheral blood, are confirmed by Schloss and Crawford. These investigators refer to the work of Goldscheider and Jacob,[6] Schulz,[7] and Bohland[8] as evidence that the fall in the leukocyte count in the peripheral blood may not be due to an actual destruction of these cells, but to a redistribution, and therefore regard this question as unsettled as yet. It should be remembered, however, as pointed out by Von Reuss,[9] that the blood cell count is very much higher during the first day than during the latter days. That is, it is higher then than during the following days when the birthweight, and correspondingly the water content of the first day, has not yet been regained. The pigment content of the intestinal discharges, and perhaps the icterus neonatorum, as well as the leukocytic destruction of the uric acid infarcts and the increased uric acid excretion, may be considered as an expression of this process.

That the increased excretion of uric acid following the increased metabolism of nucleoprotein is so well known that it is unnecessary to refer to the large literature on this subject. This fact makes attractive the view that the increased excretion of uric acid in the urine of the new-born is due, in part at least, to a destruction of leukocytes at this time, but as already noted, the fact of such a destruction seems at present to be disputed. The cause of this increase in the uric acid output must, in the light of our present knowledge, be referred in part, at least, to an increased metabolism of nucleoprotein material; but just where in the body this is taking place must be regarded as an unsettled question.

The occurrence of uric acid infarcts in the form of neutral, acid ammonium urate or a mixture of neutral and acid ammonium urate in the kidney of the new-born during the first few days of life is an established fact, and it was shown by Schloss and Crawford that uric acid infarct elements were present in all the urines of the new-born in the cases already referred to.

The purpose of the present investigation is to determine whether or not the high uric acid excretion during the first few days of life is accompanied by a simultaneous increase of this substance in the blood. While it seemed probable that this would be found to be the case, there was, nevertheless, a reasonable doubt concerning this question

5. Carstanjen, M.: Jahrb. f. Kinderh., 1900, **52,** 215.
6. Goldscheider and Jacob: Ztschr. f. klin. Med., 1894, **98,** 373.
7. Schulz: Deutsch. Arch. f. klin. Med., 1893, **51,** 234.
8. Bohland, H.: Centralbl. f. inn. Med., 1899, **20,** 361.
9. Von Reuss: Krankheiten der Neugeborenen, 1914, Springer, Berlin.

which could be settled only by actual determination of the uric acid content of the blood.

METHODS

Benedict's[10] modification of the original Folin-Denis[11] method for the determination of uric acid was tried, but found more tedious than the later codification of Myers, Fine and Lough.[12] The latter modification, with certain minor changes,[13] was used throughout the work. By this means it was always possible to obtain an unknown blue solution that was perfectly clear and easy to compare with the standard. The standard color was that made by 0.4 mg. of uric acid, and was usually diluted to 30 c.c., a dilution that gave the most satisfactory depth of color when the colorimeter was set at 20 mm. The unknown color was diluted to the volume that gave approximately the same shade as the diluted standard, the regular procedure of Myers, Fine and Lough, and much more satisfactory in our hands than the older procedure in which the dilution was more or less arbitrarily fixed, with the result that one had frequently to compare colors that were of much greater degrees of difference and therefore harder to work with.

It was noticed by Curtman and Freed[14] that Benedict's[15] standard uric acid solution slowly deteriorated, and at the end of two months was no longer standard. This is confirmed by our experience. It was customary to check the standard solution from time to time against a new solution. It was found that the standard would hold its strength for at least ten days, but that while some solutions kept for a month or more, it was unsafe to assume that a standard more than ten days old was still serviceable.

Daylight was used wherever possible in reading the colorimeter, but in a few cases this was impossible without letting the analysis stand over at some stage until the next day. While there is no evidence at our disposal that indicates that a delay in the analytical procedure vitiates the results, it nevertheless happened that in most of the few cases in which no color was obtained with the uric acid reagent, the analysis had been delayed. As to the cause of these failures, which have been previously noted by Morris,[16] we have no definite information. In order to avoid this possible source of failure it was customary to use an artificial source of light in a few analyses. This consisted

10. Benedict, S. R.: Jour. Biol. Chem., 1915, **20,** 629.
11. Folin, O., and Denis, W.: Jour. Biol. Chem., 1912, **13,** 469.
12. Myers, V. C., Fine, M. S., and Lough, W. G.: Arch. Int. Med., 1916, **17,** 570.
13. Kingsbury, F. B., and Sedgwick, J. P.: Jour. Biol. Chem., July, 1917.
14. Curtman, L. G., and Freed, M.: Jour. Biol. Chem., 1916, **28,** 89.
15. Benedict, S. R., and Hitchcock, E. H.: Jour. Biol. Chem., 1915, **20,** 623.
16. Morris, J. L.: Jour. Biol. Chem., 1916, **25,** 205.

in a 100 watt nitrogen burner of "Daylite" glass enclosed in a box having a ground glass window. It was found that pure uric acid solutions gave the same relative colorimetric readings with this light as with daylight, and that it was fully as easy to compare the colors accurately. The blue solutions produced by the uric acid of blood, however, gave readings about 0.2 mm. higher than when compared with the same standard (at 20 mm.) under exactly the same conditions with daylight. For this reason the artificial light was used only when absolutely necessary to avoid letting the analysis stand over until the next day. Not more than ten colorimetric readings in the whole number of analyses recorded in this paper were made with this light.

COLLECTION OF BLOOD SAMPLES

Blood was drawn through a sterile hypodermic needle from the superior longitudinal sinus of the new-born into a glass syringe and

TABLE 1.—DETERMINATION OF URIC ACID IN MATERNAL AND PLACENTAL BLOOD

Name	Maternal Blood, Uric Acid in 100 Gm. of Blood, Mg.*	Placental Blood, Uric Acid in 100 Gm. of Blood, Mg.	Name	Maternal Blood, Uric Acid in 100 Gm. of Blood, Mg.*	Placental Blood, Uric Acid in 100 Gm. of Blood, Mg.
Beau.	...	2.9	Wal.	3.1	3.2
Drey.	3.5	2.8	Han.	2.8	2.6
Pel.	3.6	3.3	Dahl.	2.9	3.1
McH.	2.2	2.4	Hus.	2.1	2.5
Ne.	2.9	2.7	Wes.	3.3	...
Mill.	3.1	2.8	Van.	...	3.1
Roos.	3.1	3.5	Aas.	2.1	2.3
Kr.	2.7	...	Swan.	3.7	4.1
Thom.	3.3	2.8		—	—
Han.	5.0	5.0	Average	3.1	3.1

* All figures are in milligrams per 100 gm. of blood.

expelled with force into a small weighing bottle containing 0.1 gm. of potassium oxalate.

The weights of blood samples used in the analyses recorded in this paper varied from about 9 to 18 gm., but the usual sample, particularly of infant blood, was about 13 to 14 gm.

Placental blood was collected as soon as possible after cutting the cord, which was done late, that is, after the cord had stopped pulsating. This blood, and also that of the mother, was also collected in weighing bottles containing 0.1 gm. of potassium oxalate. The maternal blood was drawn from a vein in the arm of the mother at the

TABLE 2.—Uric Acid Content of Blood of New-Born

Date of Birth	Sex	Name	Uric Acid in Placental Blood, Mg.	Uric Acid 0 to 23 Hours after Birth, Mg.	Uric Acid 24 to 47 Hours after Birth, Mg.	Uric Acid 48 to 71 Hours after Birth, Mg.	Uric Acid 72 to 95 Hours after Birth, Mg.	Uric Acid 96 to 119 Hours after Birth, Mg.	Uric Acid 5 to 8 Days after Birth, Mg.	Uric Acid 8 to 10 Days after Birth, Mg.	Weight New Born, Gm.	Water Given	Remarks
11/27/16	♂	Men.	9 hrs.	9 hrs. 4.2	.....	.....	.....	.....	.....	.....	3,445	—	1
12/ 3/16	♀	Hoff.	.....	.....	26 hrs. 3.7	.....	.....	.....	.....	.....	3,340	*	2
12/ 4/16	♀	Kah.	.....	23 hrs. 2.9	.....	.....	.....	.....	.....	.....	4,140	—	3
12/ 6/16	♂	Mart.	.....	8 hrs. 4.9	.....	.....	.....	.....	.....	10 days 1.3	3,445	*	4
12/11/16	♂	Kitt.	1.7	9 hrs. 2.7	.....	.....	.....	.....	.....	.....	3,120	*	5
12/ 9/16	♀	Rick.	3.1	.....	.....	.....	.....	96 hrs. 3.7	.....	.....	3,600	—	6
12/10/16	♂	Kenn.	1.9	.....	46 hrs. 4.0	.....	.....	.....	.....	9 days 3.5	3,375	—	7
12/16/16	♂	Boye.	3.6	.....	46 hrs. 3.8	.....	.....	.....	.....	.....	2,280	*	8 †
12/19/16	♂	Schim.	2.8	.....	44 hrs. 3.0	.....	.....	.....	.....	.....	2,900	*	9
12/16/16	♂	Berl.	.....	.....	.....	.....	.....	.....	120 hrs. 2.1	.....	3,375	—	10 ‡
12/16/16	♀	Berl.	.....	.....	.....	.....	.....	.....	120 hrs. 3.4	.....	3,230	—	11
1/21/17	♂	McDon.	.....	.....	39 hrs. 5.1	.....	.....	.....	.....	.....	4,060	—	12
1/20/17	♂	Ban.	4.3	.....	.....	.....	72 hrs. 4.1	.....	.....	.....	3,280	*	13
1/26/17	♂	Mel.	3.1	.....	.....	.....	72 hrs. 4.2	.....	.....	.....	3,980	*	14
2/ 1/17	♂	Spre.	.....	23 hrs. 2.9	.....	.....	.....	.....	.....	.....	3,160	*	15
2/ 7/17	♀	Eich.	2.1	.....	36 hrs. 3.6	.....	.....	.....	.....	8 days 1.7	2,740	*	16
2/ 9/17	♀	Thomp.	2.5	.....	.....	.....	.....	96 hrs. 2.6	.....	8 days 1.4	3,160	*	17
2/ 9/17	♂	Mor.	2.7	.....	.....	.....	.....	.....	120 hrs. 2.5	8 days 1.1	3,190	*	18
3/20/17	♂	Lars.	.....	.....	.....	.....	72 hrs. 3.8	.....	.....	.....	3,220	—	19
		Dahl.	.....	.....	.....	.....	72 hrs. 2.8	.....	.....	.....	.....	..	20
3/24/17	♂	Hol.	.....	.....	.....	48 hrs. 4.5	.....	.....	.....	.....	3,650	*	21
3/24/17	♀	Sav.	.....	.....	.....	48 hrs. 4.7	.....	.....	.....	.....	3,348	—	22
3/28/17	♂	Aas.	.....	.....	.....	48 hrs. 2.5	.....	.....	.....	.....	3,750	*	23
4/ 1/17	♀	Zimm.	.....	.....	38 hrs. 3.7	.....	.....	.....	.....	11 days 1.1	3,420	*	24
4/ 1/17	♂	Peter	.....	.....	36 hrs. 3.0	.....	.....	.....	.....	.....	3,910	—	25
4/ 4/17	♀	Hard.	.....	.....	.....	48 hrs. 3.7	.....	.....	.....	10 days 1.4	2,780	*	26 §
3/29/17	♂	Buhl.	.....	.....	.....	.....	.....	.....	.....	10 days 1.5	2,870	—	27
4/ 7/17	♀	Smi.	.....	.....	.....	.....	72 hrs. 2.7	.....	134 hrs. 3.4	.....	3,330	—	28
4/ 7/17	♀	Math.	.....	.....	.....	65 hrs. 3.4	.....	.....	.....	.....	3,020	*	29
4/ 6/17	♂	David	.....	.....	.....	.....	72 hrs. 2.9	.....	.....	.....	4,220	—	30
	♂	Peter	.....	.....	.....	.....	72 hrs. 3.7	.....	.....	.....	.....	..	31

† Premature 1 month. ‡ Twins. § Slightly premature.

time of parturition. All the new-born which served as subjects in this investigation were normal.

URIC ACID CONTENT OF MATERNAL AND PLACENTAL BLOOD

A separate series of analyses were made to determine the uric acid in maternal and placental blood in order to obtain a basis of comparison for the uric acid content of new-born blood. Several of the analyses recorded in this series had been made when it was learned that Dr. J. M. Slemons, of New Haven, Connecticut, was working on the placental blood as a separate problem. In a private communication he clearly established his priority in this matter and also reported that he had found identical values for the uric acid content of maternal

TABLE 3.—AVERAGE VALUES FOR BLOOD URIC ACID EACH DAY FROM BIRTH TO FIFTH DAY

Age	Uric Acid per 100 gm. of Blood, Mg.	Number of Analyses on Which Average is Made
At birth*	3.0	42
0 to 23 hours	3.5	5
23 to 47 hours	3.6	8
48 to 71 hours	3.9	5
72 to 95 hours	3.5	7
96 to 119 hours	3.2	2
120 to 134 hours	2.9	4
8 to 11 days	1.6	8

* This is the average value for all maternal and placental figures.

and placental blood. His figures have not yet been published, nor are they known to us.

Our figures for this series of analyses are shown in Table 1. It will be noted that the average values for sixteen determinations of the uric acid content of maternal and placental blood are identical, agreeing in this respect with the independent finding of Slemons.

Table 2 shows the results obtained with the new-born. In some cases the infants were given water (marked * in the table) in addition to their ordinary diet of breast milk. It will be noted that the content of uric acid in the blood the first three or four days after birth is higher than that of the blood of the same new-born at birth. In seven cases there is a marked decrease in this value at the end of eight to eleven days from the value obtained (in each case with the same infant) three to eight days earlier.

Table 3 shows the average values for blood uric acid each day of life from birth to the fifth day, and during the period of the eighth to the eleventh day. It will be noted that during the period between the second and third days (forty-eight to seventy-one hours) the value reaches a maximum. A much larger series of analyses would be necessary to fix beyond question the day on which this maximum average value is reached; if, on account of individual variations, this could be established at all. During the first three or four days of life this value is higher than the maternal and placental figures.

From a maximum of 3.9 mg., the blood uric acid falls off slowly to 2.9 mg. on the fifth day and then rapidly to 1.6 mg. by the eighth to eleventh day. This value agrees with the 1.3 to 1.7 mg. found by Liefmann[17] in the blood of thriving children from nine weeks to fourteen months of age, on a pure milk diet.

CONCLUSION

Our finding that there is a parallelism between the high uric acid content of the blood of the new-born and the high excretion of this substance during the first three or four days of life, is indirect evidence supporting the findings of Wells and Corper[18] and others (in opposition to the results of Schittenhelm and Schmidt[19]) that human fetal tissues possess no uricolytic power, for it would be difficult to imagine so great a production of uric acid if the tissues themselves possessed the power to destroy it. Whether the decomposition of nuclein material, which must be looked on as the cause of this uric acid increase in the blood, is related to the striking changes in the blood cells, particularly in the changes in the partition of the white corpuscles taking place at this time, or to nuclein destruction in other parts of the body, as yet unknown, or to both, must be left to the future to decide. These results fit in well, however, as the connecting link in the theoretical chain of early leukocytosis — fall in leukocytes — *flood of uric acid in the blood* — high uric acid excretion — uric acid infarcts.

We wish to thank the members of the Graduate School in the pediatric department for obtaining for us the new-born blood samples, and also the members of the obstetric staff of the University for obtaining the samples of maternal and placental blood.

Syndicate Building.

17. Liefmann, 1.: Ztschr. f. Kinderh., Orig., 1915, **12,** 227.
18. Wells, H. G., and Corper, H. J.: Jour. Biol. Chem., 1909, **6,** 321.
19. Schittenhelm, A., and Schmidt, G.: Ztschr. f. exper. Path. u. Therap., 1907, **4,** 424.

THE EFFECT ON HUMAN MILK PRODUCTION OF DIETS CONTAINING VARIOUS FORMS AND QUANTITIES OF PROTEIN *

B. RAYMOND HOOBLER, M.D.
DETROIT

The problem of constructing a diet for a mother with a failing or deficient milk supply is one of the most difficult which is presented to the pediatrist.

The laity have any number of suggestions as to foods necessary; the physician has his favorite galactogogues; the nostrum vender advertises his wares as infallible milk producers, but in spite of all of these many mothers who begin nursing their babies soon find that their babies do not thrive on the milk which they are able to produce, and though they make a desperate effort to continue breast feeding they in time fail and their babies half starved, with resistance greatly lowered, are turned over to the pediatrist to feed on some form of artificial feeding.

The literature is practically barren of any extended researches on the effect of the diet on human milk production, but several excellent pieces of work have been done on milk production of the lower animals.

Voit contributed a valuable research on a bitch.[1] The animal was fed on meat alone, meat and fat, meat and starch, starch alone, fat alone and was also starved. Professor Lusk, in summing up the result of the experiment concludes that the influence on the milk secretion was found to be comparatively small.[2] Professor Lusk studied this problem as it related itself to the fat of goat's milk. He found that starvation greatly increased the fat content of the milk which returned to its normal level when the diet was resumed.

A most illuminating piece of work has been done to increase the production of milk in dairy cows. Under the direction of F. W. Wall, E. B. Hart and G. C. Humphrey of the University of Wisconsin, much scientific data on the proper feeding of cows for milk production has been obtained and published. The economic aspect of the problem has caused them to study particularly the nutritive ratio best adapted

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* Read at the meeting of the American Pediatric Society, White Sulphur Springs, W. Va., May 28, 1917.

1. Voit: Ztschr. f. biol., 1896, 137.
2. Lusk: Science of Nutrition, 1909, p. 235.

to milk production. Hart and Humphrey, discussing this question, state that "since the time of Wolff the feeding of cows has called for a narrow nutritive ratio; that is, a high percentage of digestible proteins in the ration. The ratios vary in proportion of digestible proteins to digestible fats and carbohydrates from 1 : 4.5 to 1 : 8.5.

Not only has the quantity of the protein most suitable for milk production in cows been studied by Hart and Humphrey, but also the quality of the protein.[3] Their findings are interesting and are summed up by them as follows:

"It was found that milk proteins had an efficiency for milk production and tissue reservation of about 60 per cent., while corn and wheat grain protein showed an efficiency of 40 and 36 per cent., respectively."

Believing that the determination of these same facts as regards human milk production would be of great value in establishing a proper basis for arriving at efficient diets, this work was undertaken.

Through the cooperation of the Detroit Wet Nurses Bureau connected with the Woman's Hospital and Infants' Home, suitable healthy mothers were placed under observation.

The food was all weighed. All the urine and feces collected. The breast milk was expressed by hand and accurately measured. The water intake was recorded. The daily weight of the mothers was kept. The total nitrogen was determined on the daily urine and milk output, and on the mixed food and feces for each period. The periods were five days each with the exception of one or two which were closed at the end of the fourth day.

The diets were carefully constructed from tabulated analysis made by Atwater and Bryant.[4]

The diets used were made to vary from a narrow nutritive ratio, that is, 1 : 4 to a very wide nutritive ratio, that is, 1 : 15. These ratios refer to the proportion of digestible protein to digestible carbohydrates and fats, the latter reduced to a cardohydrate basis.

Not only were diets with varying ratios used, but diets were also fed in which different types of protein predominated: In certain diets meat protein was used, in others, milk, cereal and nut protein. Some of these diets were high in calories, others, medium and low.

The diets may be characterized as follows:

Diet No. 1: Calories 3,767; nutritive ratio 1 : 5; fat to carbohydrate 3 : 2, predominating in animal protein, especially milk. Sources of protein as follows: milk 33 per cent., meat 21.5 per cent., cereal 33.5 per cent., vegetable 11.5 per cent.

Diet No. 2: Calories 3,446; nutritive ration 1 : 8; fat to carbohydrate 2 : 3, animal and vegetable protein in equal quantities: meat 27.7 per cent., milk

3. Hart and Humphrey: Jour. Biol. Chem., **302,**

4. Atwater and Bryant: U. S. Dept. Agriculture, Bull. 28

14.5 per cent., eggs 8.5 per cent., vegetable 19.8 per cent., cereals 18.1 per cent., nuts 9.4 per cent., fruits 2 per cent.

Diet No. 3: Calories 3,503; nutritive ratio 1:9; fat to carbohydrate 1:4; vegetable protein entirely with exception of milk: cereal 60.5 per cent., vegetables 14.2 per cent., fruit 1.4 per cent., milk 23.9 per cent.

Diet No. 4: Calories 3,634; nutritive ratio 1:15; fat to carbohydrate 2:1; animal and vegetable protein in equal quantities, as follows: milk 37.1 per cent., meat 10.5 per cent., cereals 28.3 per cent., vegetable 18.6 per cent., nuts 5.6 per cent.

TABLE 1.—DIET 9

Food	Amount	Food	Amount
Oatmeal	150 gm.	Cocoa	4 gm.
Bread	200 gm.	Apple sauce	50 gm.
Butter	25 gm.	Lima beans	100 gm.
Round steak	150 gm.	Sugar	50 gm.
Eggs	100 gm.	Cream 20%	100 gm.
Potatoes	200 gm.	Spinach, squash or carrots	100 gm.
Milk	500 gm.	Hypophosphites	½ oz. three times a day before meals
Rice custard	100 gm.		

This diet approximated 2,927 calories. It contained 130 gm. of protein, yielding 20.64 gm. of nitrogen. The sources of the protein were as follows: meat 40.5 per cent., milk 15.7 per cent., eggs 10.5 per cent., cereal 21.2 per cent., vegetables 12.1 per cent.

Diet No. 5: Calories 2,088; nutritive ratio 1:9; fat to carbohydrate 1:1; animal protein diet, meat predominating: meat 32.9 per cent., eggs 12.9 per cent., vegetable 19.8 per cent., cereals 14.8 per cent.

Diet No. 6: Calories 2,774; nutritive ratio 1:6; fat to carbohydrate 3:2; animal protein predominating: meat 40.5 per cent., milk 15.7 per cent., eggs 10.5 per cent., cereal 21.2 per cent., vegetable 12.1 per cent.

Diet No. 7: Calories 2,842; nutritive ratio 1:5; fat to carbohydrate 1:1; animal protein predominating: meat 40.5 per cent., milk 15.7 per cent., eggs 10.5 per cent., cereal 21.2 per cent., vegetable 12.1 per cent.

TABLE 2.—NUT DIET 10

Food	Amount	Food	Amount
Malted nuts	100 gm.	Chocolate	8 gm.
Protose (nut product)	150 gm.	Bread	200 gm.
Pecans	25 gm.	Potatoes	200 gm.
Almonds	25 gm.	Lima beans	100 gm.
Walnuts	25 gm.	Peas	200 gm.
Peanut butter	50 gm.	Bananas	100 gm.
Cocoa	8 gm.	Apple sauce	50 gm.

This diet contained approximately 2,670 calories. The ratio of protein to fat and carbohydrate (all reduced to basis of calories) was 1:5. The actual number of grams of protein ingested was 104.68, yielding 16.75 gm. of nitrogen. Of this intake, 89 per cent. was absorbed. The mothers took the diet readily and enjoyed it.

Diet No. 8: Calories 2,286; nutritive ratio 1:13; fat to carbohydrate 1:4; pure vegetable protein: cereal 54.2 per cent., vegetable 40.3 per cent., fruit 5.5 per cent.

Diet No. 9: Calories 2,927; nutritive ratio 1:4; fat to carbohydrate 1:1; animal protein predominating: meat 40.5 per cent., milk 15.7 per cent., eggs 10.5 per cent., cereal 21.2 per cent., vegetable 12.1 per cent., hypophosphites.

Diet No. 10: Calories 2,670; nutritive ratio 1:5; fat to carbohydrate 1:1; pure vegetable diet, nut protein predominating: nuts 66.2 per cent., vegetable 17.7 per cent., cereal 14.8 per cent., fruits 1.3 per cent.

Diet No. 11: Calories 4,285; nutritive ratio 1:20; corn products diet with gelatin, being an amino-acid deficient diet.

Diet No. 12: Same as Diet 11 with casein added, making the diet an amino-acid sufficient diet.

Exact quantities used in Diets 9 and 10 are given.

One of the questions which by the use of these diets we sought to answer was this: Was there any difference in the quantity of milk protein produced when mothers were fed on a wide or a narrow nutritive ratio?

Tables 3-7 are submitted as an answer to this question:

TABLE 3.—NITROGEN BALANCE SHEET ON ANIMAL PROTEIN DIET

Diet No.	Nutritive Ratio	Nitrogen, Grams in 24 Hr.						Name of Mother
		Intake	Feces	Absorbed	Urine	Milk	Balance	
9	1:4	20.64	1.63	19.01	9.54	2.63	+6.81	Ruth
9	1:4	20.64	1.60	19.04	9.79	2.23	+7.01	Grace
7	1:5	16.56	1.68	14.77	10.94	2.88	+1.04	Ruth
7	1:5	16.56	1.05	15.51	13.35	2.12	+0.034	Grace
1	1:6	19.60	1.83	17.76	9.45	2.88	+5.10	Ruth
1	1:6	19.60	1.53	18.06	10.92	2.35	+4.79	Grace
2	1:8	16.11	1.68	14.45	11.45	2.28	+0.71	Ruth
2	1:8	16.11	1.60	14.51	16.55	2.19	—4.24	Grace
6	1:6	14.68	1.66	13.01	12.70	2.39	—2.08	Ruth
6	1:6	14.68	0.98	13.69	14.12	1.68	—2.11	Grace
6	1:6	13.94	1.38	12.56	6.32	1.26	+4.72	Mary
6*	1:6	14.75	0.231	14.52	5.22	1.62	+7.66	Mary

* Diet same as No. 6 with the addition of 250 c.c. cow's milk.

It will be seen that when diets were given containing a narrow nutritive ratio the actual number of grams of nitrogen excreted in the milk was greater than when fed on a wider nutritive ratio. Not only was this true, but the mothers were in positive nitrogen balance on the narrow ratios, while on the wider ratios they went into negative balance and were forced to give up some nitrogen to keep up the milk production, thus "milking the flesh off of their backs." These facts hold

true only when the protein in the diet is from animal source. It is difficult to keep the mothers in positive nitrogen balance regardless of how narrow a ratio is fed when given vegetable protein, as is shown by Table 4.

TABLE 4.—NITROGEN BALANCE SHEET ON VEGETABLE PROTEIN DIET

Diet No.	Nutritive Ratio	Nitrogen, Grams in 24 Hr.						Name of Mother
		Intake	Feces	Absorbed	Urine	Milk	Balance	
10	1:5	16.75	1.68	15.07	12.99	2.79	—0.711	Ruth
10	1:5	16.75	1.67	15.08	16.77	2.50	—1.69	Grace
3	1:9	12.71	1.46	11.32	8.76	2.46	+0.093	Ruth
3	1:9	12.71	1.68	11.03	9.75	1.84	—0.559	Grace
8	1:13	6.09	1.77	4.32	8.77	2.45	—6.90	Ruth
8	1:13	6.09	2.15	3.94	7.32	2.04	—5.38	Grace

It will be noted that there is a marked difference, viz., the mothers were thrown into negative nitrogen balance even when fed on a narrow nutritive ratio when the protein supplied was of the vegetable type.

This leads us to the second question which these observations were designed to answer: What type of protein is best adapted to the production of milk protein? What has been shown in Table 4 is much more convincingly shown in Tables 5 and 6.

Table 5 shows that the mothers, when fed on diets predominating in animal protein, remain in positive nitrogen balance as long as the nutritive ratio is 1:6 or narrower, and that above that they go into negative balance.

Table 6 shows the inadequacy of diets in which there is vegetable protein alone or in which it predominates. In Diets 8 and 10 there was vegetable protein only, which resulted in negative nitrogen balance, while in Diet 3 the single animal protein milk added to a vegetable diet was able to establish nitrogen equilibrium.

In estimating the efficiency of a diet one should take into consideration not only the amount of nitrogen excreted in the milk, but also the amount of nitrogen added to or withdrawn from the tissues of the mother. These two factors when considered in relation to the nitrogen absorbed gives an efficiency index.

Tables 5 and 6 show the efficiency of the various diets when considered from this standpoint.

It will be noted that the diets are arranged according to nitrogen efficiency and vary from 49 per cent. positively efficient to 100 per cent negatively efficient. It will be noted that diets 1, 7 and 9 have a narrow nutritive ratio and are made up of two-thirds or more of

animal protein. These diets give the best protection to the tissues of the mother. It will also be noted that the diet containing 3,767 calories (overfeeding) did not give as good a result as the diet containing 2,928 calories (overfeeding is not necessary).

TABLE 5.—NITROGEN DISTRIBUTION AND EFFICIENCY TABLE ON ANIMAL PROTEIN DIET

Diet No.	Nutritive Ratio	Food Calories	Milk Produced, C.c.	Milk, Calories	Percentage of Nitrogen Intake Found In					Name of Mother
					Milk	Feces	Urine	Retained	Efficiency	
9	1:4	2,927	1,522	968	12.7	7.9	46.3	+33.0	49.6	Ruth
9	1:4	2,927	1,630	1,192	10.8	7.7	47.4	+33.0	48.5	Grace
7	1:5	2,842	1,486	962	17.3	10.9	66.0	+ 5.6	26.4	Ruth
7	1:5	2,842	1,751	967	12.8	6.3	80.4	+ 0.3	13.9	Grace
1	1:6	3,767	1,475	1,042	14.7	9.3	48,5	+27.0	43.0	Ruth
1	1:6	3,767	1,445	1,071	12	7.8	55.7	+24.0	38.8	Grace
6	1:6	2,774	1,329	1,000	16	11.3	85.6	—13.3	2.3	Ruth
6	1:6	2,774	1,521	1,269	11	6.7	96.2	—14.3	—3.0	Grace
6	1:6	2,388	523	.....	9.05	9.9	45.0	+32.1	47.6	Mary
6*	1:6	2,560	666	.....	11.03	0.09	35.4	+52.0	74.2	Mary
6†	1:6	2,411	813	.....	26.4	10.9	58.4	+4.3	34.3	Mary
2	1:8	3,446	1,425	835	14.2	10.3	71.0	+4.3	20.7	Ruth
2	1:8	3,446	1,375	698	13.6	10.0	102.0	—26.4	—1.4	Grace

* Diet same as No. 6 with the addition of 250 c.c. fresh cow's milk.
† Diet same as No. 6 with the addition of 50 gm. malted milk.

TABLE 6.—NITROGEN DISTRIBUTION AND EFFICIENCY TABLE ON VEGETABLE PROTEIN DIET

Diet No.	Nutritive Ratio	Food, Calories	Milk Produced, C.c.	Milk, Calories	Percentage of Nitrogen Intake Found In					Name of Mother
					Milk	Feces	Urine	Retained	Efficiency	
10	1:5	2,670	1,758	1,006	16.6	10.0	90.0	—4.2	13.8	Ruth
10	1:5	2,670	1,620	1,372	14.9	10.0	90.0	—10.9	5.3	Grace
3	1:9	3,503	1,370	824	19.3	11.0	88.0	+0.22	22.0	Ruth
3	1:9	3,503	1,174	766	14.4	13.0	86.8	—4.38	11.0	Grace
8	1:13	2,286	1,507	786	40.2	29.0	143.0	—113.0	—102.8	Ruth
8	1:13	2,286	1,586	973	32.8	35.0	120.0	—87.0	—8.6	Grace
4	1:15	3,634	1,362	970	27.6	26.3	95.8	—49.9	—30.2	Ruth
4	1:15	3,634	1,416	1,059	22.1	18.3	102.0	—42.4	—2.2	Grace

In Diets 3, 4 and 10, in which vegetable protein exceeded the animal protein, the efficiency was much diminished, though still sparing the maternal tissues. Special consideration should be given to Diet 10,

since it was a purely vegetable diet, 60 per cent. of its protein being derived from nut foods. The tissues were amply protected, and a large quantity of milk was produced containing more nitrogen and more calories in twenty-four hours than any other diet used in this series.

Diet 6 was just able to maintain nitrogen equilibrium, although nitrogen excreted in the milk was considerably lessened.

A diet which would just maintain nitrogen equilibrium is in many respects preferable to one which tends to add weight, many mothers complaining of this increase in weight during the nursing period. Diets 4, 5 and 8 put the mothers in negative balance. It will be noted that the ratios were wide, and in Diets 5 and 8 the calories were very low. These two diets were constructed so as to approximate diet lists secured from a number of mothers whose milk supply was failing. It is evident why a mother receiving only from 6 to 8 gm. of nitrogen a day cannot continue to give up from her own tissues an equal quantity of nitrogen, although nature will go to extremes in maintaining a milk supply.

The results obtained from a pure vegetable diet (Diet 8) suggest the futility of excluding from the diet at least a certain amount of animal protein. Many mothers are trained during the latter part of pregnancy to eliminate albuminous food from their dietary because of albuminuria. The fear that the albuminuria may persist during the puerperium causes many mothers to continued a low protein diet, which frequently is the cause of failing milk supply.

The question as to what form of animal protein is best adapted to the production of milk protein is an important one. There is considerable evidence that cow's milk protein, while not an essential part of a diet, contributes considerably more than its mere caloric value in increasing not only the protein output in the milk, but also aids greatly in protection of the maternal tissues from destruction. This is shown in results obtained in Diet 3, which was made up entirely of vegetable protein with the exception of milk which consisted of 23.5 per cent. of the protein fed. This small amount of animal protein was sufficient to keep the mother in positive nitrogen balance and produce more calories (1,590), and more milk nitrogen (2.46 gm.) in the milk produced than Diet 2, which contained 50 per cent. animal protein, a small proportion of which was derived from milk. Hart and Humphrey found that milk protein when fed to cows had an efficiency for milk production and tissue preservation of about 60 per cent. in dairy cows, while corn and wheat protein had an efficiency of 40 per cent. and 36 per cent., respectively.

The effect of milk protein on tissue preservation as well as its effect on milk nitrogen is shown in Table 7.

TABLE 7.—EFFECT OF MILK PROTEIN ON TISSUE PRESERVATION AND MILK NITROGEN

Diet No.	Nitrogen Absorbed Four Day Period	Nitrogen in Milk	Nitrogen in Milk of Tissue Formed or Destroyed	Nitrogen Efficiency
6	50.242	5.04	23.949	47.60
6*	58.082	6.48	37.176	74.2

* 250 c.c. of fresh cow's milk added to Diet 6.

From Table 7 it will be seen that by the addition of 8 ounces of milk daily to a diet previously poor in milk protein the efficiency of such diet was raised from 47.6 per cent. to 74.2 per cent., and that the nitrogen of the milk was raised from 5.04 gm. to 6.48 gm.

SUMMARY

1. A nutritive ratio of 1:6 or narrower seems best adapted to the need of nursing mothers. (This ratio refers to the proportion of digestible protein to digestible fat and carbohydrate, the latter reduced to a carbohydrate basis.)

2. Animal protein is more suitable than vegetable protein in supplying nitrogen for milk and maintenance of nitrogen balance.

3. The protein derived from nuts when fed with other vegetable protein is suitable for supplying milk protein and for maintaining nitrogen equilibrium.

4. A diet composed exclusively of cereals, fruits and vegetables does not supply sufficient protein for elaborating milk protein and causes a severe drain on tissues of mother.

5. Of the various forms of animal protein, that which is derived from cow's milk, seems particularly suitable for the production of human milk protein, as well as for the preservation of maternal tissues.

CONGENITAL SKIN DEFECTS*

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The so-called congenital skin defects have not been recorded frequently in the literature. The majority of the accessible reports deal with skin defects of the scalp, which vary in size from a pinpoint to a small coin. They are usually circular in shape, sometimes oval or irregular, and generally have clean-cut edges.

The first authentic case seems to have been recorded by an English physician, Campbell, who in 1826 reported a skin defect of the scalp the size of a crown, which assumed the characteristics of a progressive ulcer. Priestley, in 1859, reported a congenital skin defect over the anterior fontanel about the size of a shilling. The defect healed in a short time, the process of repair beginning at the edge of the ulceration. Billard, in 1828, reported a case of skin defect over the parietal bone supposed to have been caused by a uterine polyp of the mother. Emanuel, in 1905, reported an infant with congenital absence of skin involving hands, legs and feet. The child was born dead. There was no evidence of syphilis. Numerous reports have been published from time to time which will be referred to in tabulated form and in the references at the end of the article.

Most writers on the subject of congenital defects attribute them either to errors in development, or to inflammatory adhesions between the external layers of the skin and the amnion. At the point where this adhesion has taken place, the growth of flat epithelium is retarded. As the liquor amnii accumulates, the so-called Simonart's bands are formed. These are adhesions which form threads or bands between the amnion and the fetus. If these bands are torn loose from the integument of the fetus, a skin defect remains. Not infrequently, these skin defects are associated with other congenital malformations, for instance, with deformities of the extremities such as deficient fingers, hands, or feet.

As has already been noted, the lesions are found most frequently on the scalp. They may occur, however, on the trunk, though in one case reported by Bürger, in addition to the scalp defect, ulcerating lesions occurred on both knees and on both elbows. Macé reported an infant who at the time of birth showed these characteristic defects

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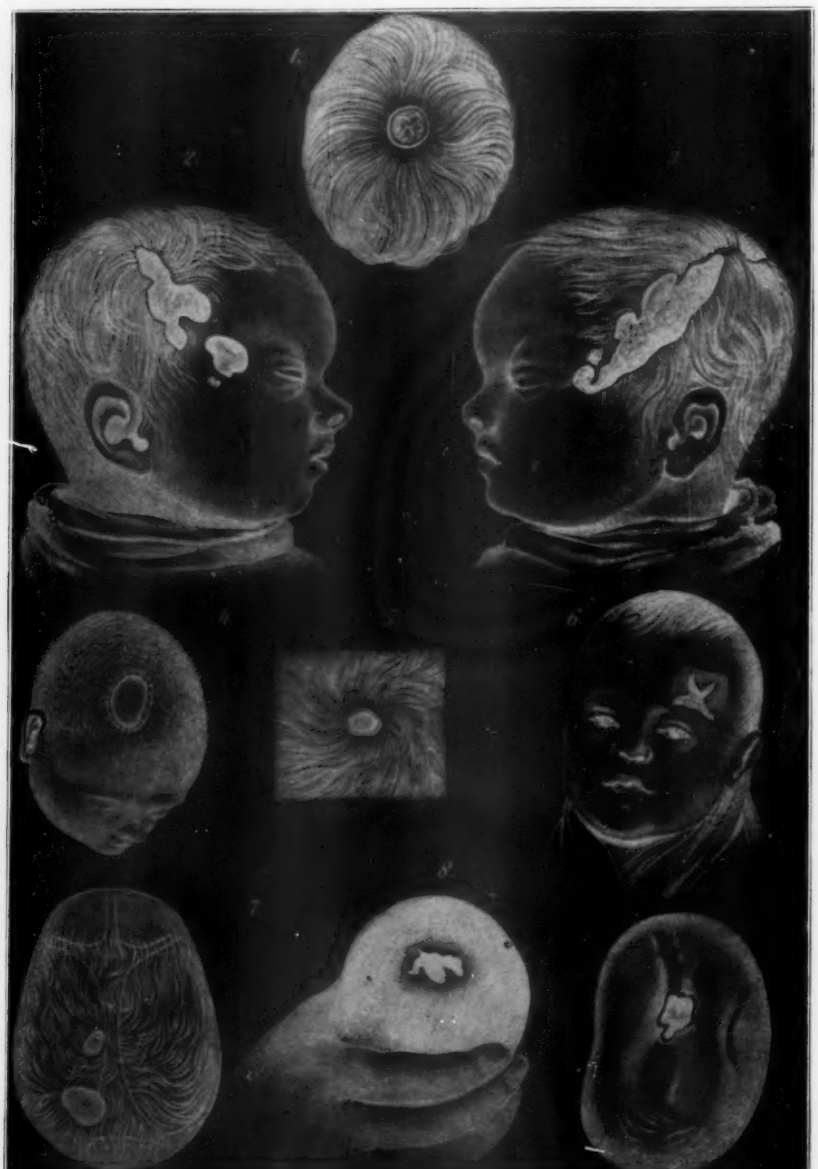

Fig. 1.—Reported cases of skin defect of the scalp. 1, Priestley's case; 2 and 3, von Hebra's case; 4 and 5, von Hofmann's; 6, Dohrn's; 7, Bonnaire's (3d), 8, Bonnaire's (4th); 9, Mathe's (1st). See accompanying table.

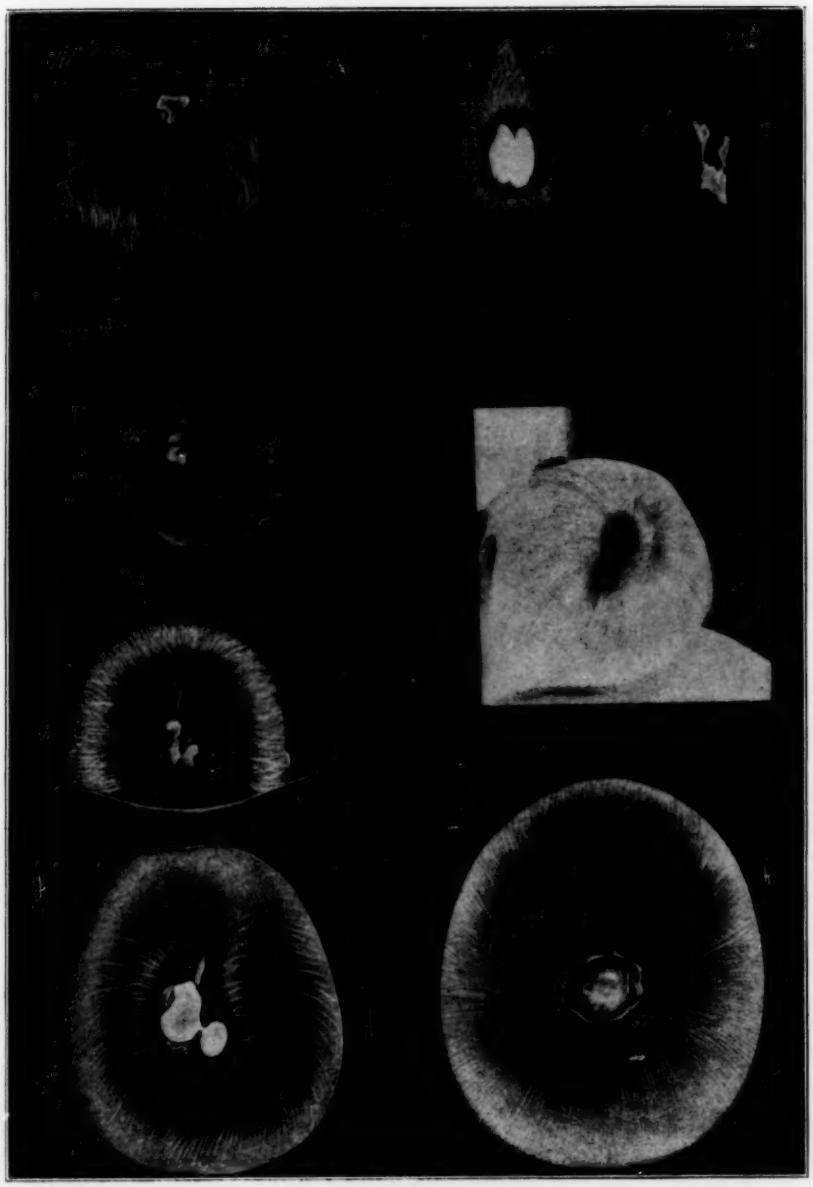

Fig. 2.—10, Mathe's (2d) case; 11, Dittrich's (1st); 12, Dittrich's (2d); 13, Schultze's; 14, von H. Vörner's; 15 and 16, Winckel's; 17, Keller's case. See accompanying table.

on the right buttock. This lesion was about 3 cm. in circumference, bled readily, and had a red periphery. The same author reported another infant with a congenital, ulcerating skin defect on the right forearm and a cicatrized defect on the left. The ulcer healed promptly. Emanuel's report, previously mentioned, refers to congenital skin defects occurring on hands, legs, feet, over the temples and the bridge of the nose, and also an area in the middle of the lumbar region.

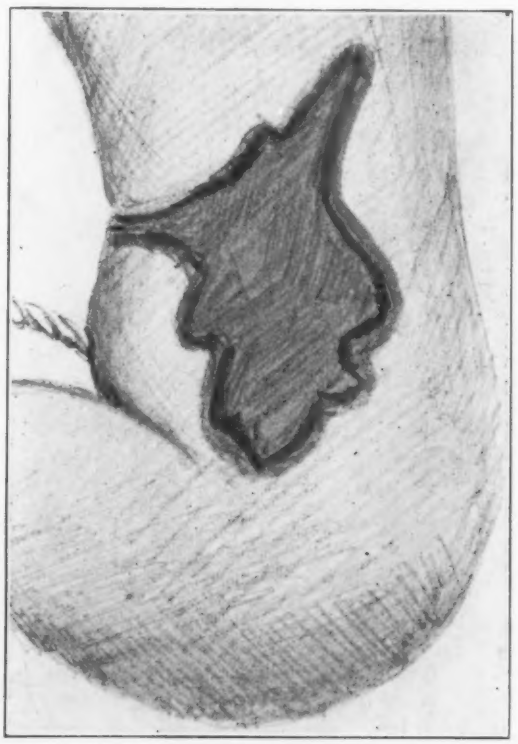

Fig. 3.—Hahn's case showing one side of a bilateral skin defect on lateral portion of the trunk.

The defects are usually visible at birth. They seem to bear no relation to trauma during parturition, and undoubtedly originate *in utero*. Occurring singly or in groups, they present ulcerated areas which affect both layers of the skin. The subcutaneous tissues are not involved. The ulcerated areas, as in my case, are sharply defined, somewhat irregular in outline; the base of the ulcer is red and secretes a thin lymph.

Of the histologic examinations made in these cases, Keller found that minute hairs occurred in the affected area. It should be remarked,

however, that this finding is contrary to other histologic reports. In some of the recorded cases, the area involved developed hairs later in life. Microscopic examination not only shows the failure of the epidermis to develop, but also, that the skin glands, if they develop at all, do so in a rudimentary manner. It is worthy of note that the microscopic examinations fail to show any old or recent degeneration in the disease area or in the surrounding tissue. This would tend to exclude a secondary process due to some intra-uterine inflammation. It is noteworthy that microscopically the area under examination shows the absence of epithelial structures, smooth muscle and adipose tissue.

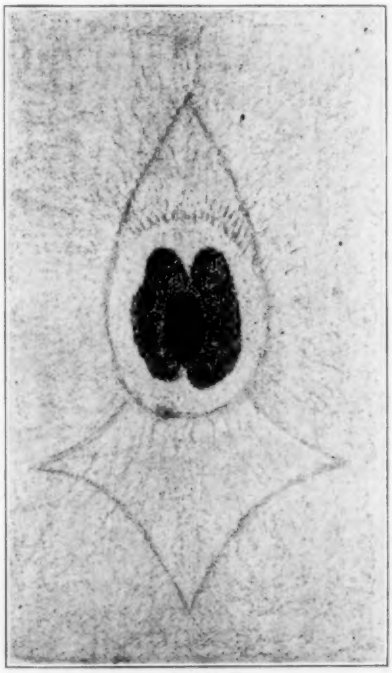

Fig. 4.—Dittrich's case of skin defect of the scalp.

Because of the well developed connective tissue, one is led to assume that this defect of skin can be traced to the early period of embryonal life. Because the lesions occur early in fetal life, the hair buds do not develop and the sebaceous glands fail to appear or are rudimentary. That the defects are not due to an injury is shown by the fact that they are already present in early fetal life. The condition has been referred to as a congenital atrophy of the skin, but this can hardly apply for the reason already mentioned, namely, that atrophy implies degeneration, and no degenerative process can be demonstrated. There is an abundance of connective tissue present in

CONGENITAL SKIN DEFECTS OF THE NEW-BORN

Reporter and References	Sex and Age	Localization and Character of Defect	Remarks
Campbell: Edinburgh Jour. Med. Sc., 1826, **5,** 82.	New-born	Parietal; circular defect, 3¾ cm. in diameter; ulcer with progressive characteristics	.....
Same as above	New-born	Parietal; scarry ulcer	.....
Billard: Traité d. mal. d. enf., New Series, Paris, 1828, p. 232.	New-born	Left parietal; about 1½ in. by ⅓ in. defect	There was a polyp involving uterus of mother, which caused destruction of part of uterine wall.
Abele: Med. Cor.-Bl. d. Wurten. ärzt. Verein, 1835, **5,** 1.	New-born	Upper half of left frontal region; 1½ by 1 inch wide; skin defect	Trauma; mother injured 10 weeks before.
Dieterich: Würtemb. Kor.-Bl., 1838, p. 5.	New-born	Round defect, 1¼ cm. in diameter; granulated; scar 1 by 1½ inches; frontal region	Trauma; mother injured 10 days before birth.
Hahn: Jour. d. Chir. u. Augenh., 1840, **30,** 156.	New-born	Large irregular defects on right and left side of trunk; almost symmetrical; defects united above navel	.....
Priestley: Trans. Obst. Soc. London, 1859, **1,** 60.	New-born	Circular defect, 2¼ cm. in diameter, of small fontanel; healing beginning at edge	.....
Schatz: Monatsschr. f. Geburtsh., 1869, **34,** 110.	10 days old	Right side of head; 7½ cm. long; skin erosion	Pelvic exostosis; possibly the erosion was due to injury by finger nail of nurse.
Tarnier: Union méd., 1872, **33,** p. 391.	1 day old	Parietal; covered by fine scar	.....
v. Hebra: Mitteil. d. Wien. embryol. Inst,. 1882, **2,** 85.	Full term	Both sides of head, 6 cm. by 4 cm. and 12 mm. by 5 cm., respectively; long, thin strips of defect; serous fluid beneath	Definite development of connective tissue.
v. Hofmann: Wien. med. Presse, 1885, No. 18; also Lehrb. d. gericht. Med., 1895, Part 2, p. 776.	4 mos. fetus	Large fontanel; 1 cm. size; granulated defect	Author considers these cases due to Amniotic adhesions (Amniotische Bände).
Same as above	Female, full term	Parietal; 5 to 6 cm.; glossy appearing defect	
Same as above	Male, full term	Parietal; circular defect; 4 cm. in diameter	
Dohrn: Ztschr. f. Geburtsh. u. Gynäk., 1888, **14,** 366.	New-born	Left frontal region; crusted strip, 1 cm. long; red granulation; excorciated	.....
Bonnaire: Progès méd., 1891, **13,** 481 and 497.	Two full-term children	Sagittal suture; defect 3 cm. in diameter; gelatinous surface	Microscopically examined; thinks due to amniotic bands or adhesions.
Same as above	7½ mos. fetus	Small fontanel circular defect, 1¼ in. in diameter size; pale covered defect; 2 smaller defects; one in sagittal suture; one in occipital region	Microscopically examined; deformed in several ways; hydrocephalus, hydramnion, etc.; amniotic bands, author thinks cause.
Same as above	One of full-term twins	Middle of sagittal suture; 12 mm. diameter; gelatinous surface	Amniotic bands author thinks cause.
Same as above	Full term child	L. os parietal; midline small bean-sized ulcer; scar tissue (hydramnion case)	Microscopically examined; same cause as above
Schrader: Zentralbl. f. Gynäk., 1893, **17,** 374.	Full term	7 cm. by 1 cm. left of parietal; blood crust	Hydrocephalus externus; died 3 months later.
Mathes: Inaugural Diss. Marburg, 1894.	25 cm. long fetus	Parietal; 6 by 8 cm. size defect with glazed surface	Microscopically examined; author thinks cause is amniotic adhesions.
Same as above	Full term child	2 cm. in front of small fontanel; irregular triangular; granulated surface	

CONGENITAL SKIN DEFECTS OF THE NEW-BORN—(*Continued*)

Reporter and References	Sex and Age	Localization and Character of Defect	Remarks
Dittrich: Vierteljahr. f. gericht. Med., 1895, **9**, 258.	New-born	Parietal; 20 by 17 sq. mm.; blackish skin defect	
Same as above	?	Middle of sagittal suture 2.5 by 1.5 cm. defect; 2 smaller defects over the small fontanels	Histologically examined.
Schultze: Ztschr. f. Geburtsh. u. Gynäk., 1895, **31**, 225.	Full term	Left frontal bone, irregular spur with skin injury; similar behind external ear, 1 cm. size.	Bone injury due to passage of head in the promontory; defect due to amniotic band.
Volkmann: Berl. klin. Wchnschr., 1898, **35**, 1025.	Full term	L. parietal; part of small fontanel; 2 bean-sized defects; granulated	Author thinks cause is amniotic adhesions.
Weindler: Zentralbl. f. Gynäk., 1899, **23**, 414.	3 weeks old child	Small fontanel; circular defect, 1¾ cm. in diam.; hard defect with granulated surface; flat edges	Author thinks cause is amniotic adhesions.
Alain: Jour. méd. de Bordeaux, 1901, **31**, 388.	Full term	2 cm. in font of large fontanel 7 sq. cm. size defect; smaller defect to the left	.....
Macé: Bull. Soc. d'obstétrique, Paris, 1901, **4**, 8.	New-born	Circular defect 3 cm. in diam. on buttock; sphacelic plaque surrounded by inflammation	.....
Same as above	New-born	Ulceration 3 cm. by 5 cm. on right forearm; same, but cicatrized on left forearm	.....
Vörner: Arch. f. Dermat. u. Syph., 1903, **66**, Part 3, p. 407.	4 year old boy	Defects on right and left side of parietal, one about 25 sq. mm., the other 15 mm. in size	Histologically examined aplasia.
Bürger: Zentralbl. f. Gynäk., 1903, **27**, 644.	Full term (13 days old child)	Parietal, rayed scarry defect; defects also on both knees and both elbows (excoriation or ulceration)	Author thinks amniotic adhesion developed from an abdominal injury received by mother in 4th month of pregnancy.
Müller: München. med. Wchnschr., 1903, **68**, 1065.	Female, 32 yrs.; defect since birth	Pronounced erythema pudoris on chest, neck and face; spots size of quarter	Due to faulty development of skin, causing innervation of blood vessels.
Same as above	Male, 48 yrs.; defect since birth	Defect similar to above	Due to same cause as above.
Winckel: Samml. klin. Vortr., 1904, Nos. 373 and 374.	Full term	Small fontanel; suppurative defect	Harelip, cleft-palate, etc.
Emanuel: Report Soc. for Study Dis. of Child., 1905-1906, **6**, 157.	Stillborn at full term	Absence of skin on hands, legs, feet, over temples, nose, middle lumbar region	Arrest of development of layers of epidermis.
Keller: Vierteljahresschr. f. gericht. Med., 1908, **35**, 223.	6 mos. fetus; 25 cm. long	Defect on right side of head; skin bluish-red; 1 sq. cm. size defect; mainly in parietal region in sagittal suture	Fetus extracted manually after dilatation; uterine hemorrhage; author thinks defects due to amniotic adhesions.
Liedig: Ztschr. f. Med.-Beamte, 1908, **21**, 607.	Full term	Circular defect 6 mm. in diam. on head	Occurred in utero.
Schulte: Deutsch. med. Wchnschr., 1909, **35**, 86.	24 hour old	On left parietal bone and sagittal suture above small fontanel; 6 by 5 cm.	.....

these lesions, and consequently the defect resembles hypertrophy more than atrophy.

If one investigates the recorded cases, it is found that these defects have been observed both in stillborn children and those who died shortly after birth, as well as in normal children who survive. Unless the skin defect is wide in extent, there is no apparent reason why the slight malformation should of itself be incompatible with the life or the general health of the infant. The local skin condition is sufficient to explain the failure of hair follicles and sebaceous glands to develop. The majority of lesions tend to cicatrize in a short time, thus providing an adequate covering for the denuded surface.

From the standpoint of differential diagnosis, it may be thought that possibly these lesions are due to trauma. We have already pointed out that there is no inflammatory reaction and no degenerative process in the region of the defects. In the case which came under my observation, the lesions were round and symmetrical, and, as in most of the reported cases, the birth was normal.

REPORT OF CASE

The case which has led to this brief clinical report was a newly born infant that was referred to me by Dr. J. B. DeLee of the Chicago Lying-In Dispensary. The mother had previously borne four perfectly normal children. Both parents were in good health. The labor was normal and the baby was normal in weight and size. It presented a defective skin area over each knee. These areas are well shown in the accompanying colored drawing (Fig. 1). They were about 1 by 1¼ inches in area; they were symmetrical, that is, almost identical in size and shape; they seemed to be, so to speak, irregularly punched out. The skin around the edges was drawn and puckered, presenting the appearance of ulcers with irregular edges. The base was beefy red and moist. A few small foci on this red, ulcerated surface showed a yellow exudate. A whitish scar seemed to cross the ulcerated area, appearing like a connective tissue band, which indicated the beginning of cicatrization. It may be said that from week to week the ulcers gradually covered over, so that after five or six weeks the denuded, red areas were replaced by shiny, white patches, somewhat lighter in color than the surrounding skin. The healing process was complete, and the baby appeared normal in every respect.

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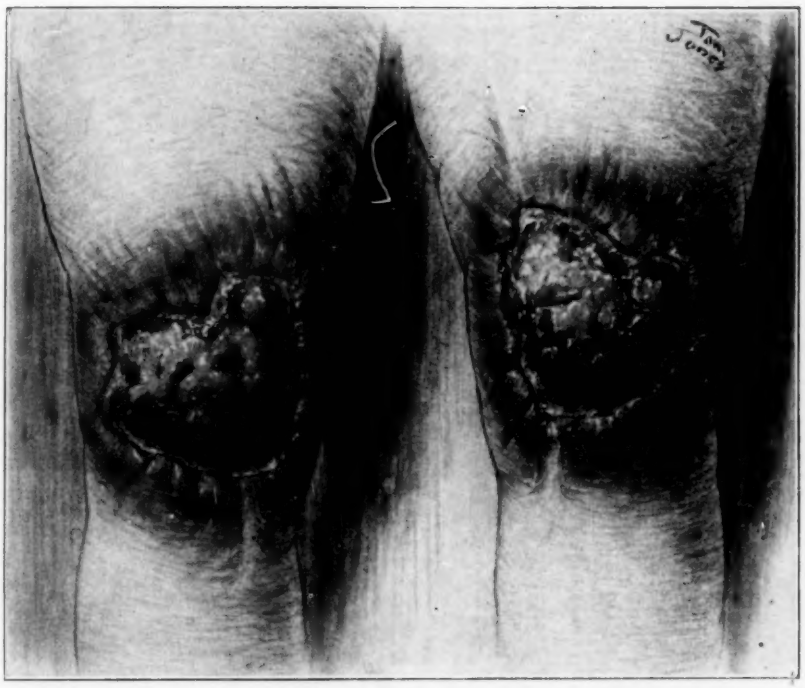

Fig. 5.—Author's case of bilateral skin defect of the knees.

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PROGRESS IN PEDIATRICS

REVIEW OF POLIOMYELITIS*

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HISTORICAL

In the middle of June, 1916, announcement was made of a group of cases of infantile paralysis in Brooklyn, New York.[1] During the summer the disease assumed the proportions of a great epidemic which, in its severity and extent of territory involved, is probably unprecedented. There were 24,000 cases in the United States.[2] About one-half of these were reported in the state of New York. In New Jersey, Connecticut, Massachusetts, Minnesota, Delaware, Rhode Island and Pennsylvania, the epidemic was also extensive. In four states, no cases were reported. These were Nevada, New Mexico, Georgia and California.[3]

EPIDEMIOLOGY

The epidemiologic characteristics of this disease have been the subject of numerous reports, the sum of which not only conflict with the conception of the disease presented from an experimental aspect, but do not harmonize with generally accepted ideas regarding other infectious diseases.

Various observers have differed regarding its mode of transmission, some favoring the human contact theory, others favoring the "place infection" theory, thereby incriminating the food supply, domestic animals, insects or other local environmental factors in the transmission of poliomyelitis.

Frost[4] summarizes these data. Epidemics offer unexplainable irregularities in their geographic distribution. While most available data suggest that the disease distributes itself from place to place along avenues of public travel, there are notable exceptions. The epidemic which occurred in New York City in 1907 did not extend to Phila-

* Submitted for publication July 1, 1917.

1. Medical News: Jour. Am. Med. Assn., 1916, **66,** 2076.
2. Emerson, Haven: Bull. Johns Hopkins Hosp., 1917, **28,** 131.
3. Lovett, R. W.: Jour. Am. Med. Assn., 1917, **68,** 411.
4. Frost, Wade H.: U. S. Public Health Service, Hyg. Lab. Bull. 90; U. S. Public Health Reports, Reprint 350.

delphia despite its close communication. In 1910, a severe epidemic occurred in Washington, D. C., and a smaller outbreak in Philadelphia, while Baltimore, lying between these two cities, escaped with only a few cases. Such instances lend support to the theory of infection by other factors than the human carrier, such as an infection of local water or food supply, or one carried by insects or domestic animals. On the other hand, the rapid extension of the disease along foci radiating from the original source of infection is in favor of the "human traffic" idea, rather than that of "place infection." This irregularity of distribution finds an analogy in the transmission of cerebrospinal meningitis, a disease which appears to be directly contagious. The maximum prevalence of the disease in the summer is a rather constant characteristic which has been used as an argument against its being carried by contact infection, since in this respect it is unlike the other contagious diseases, so carried, whose maximum prevalence is in the winter months. On the other hand, Frost states that "cases have occurred during the last few years in every month of the year." This constant presence of the disease disproves the hypothesis that a new epidemic may be ascribed to the introduction of a new infection.

The small total incidence of the disease has been a puzzling feature in its study. It has been further observed that in such an affected locality, the epidemic will not likely recur for at least two years.

In rural communities the incidence of the disease has been greater than in thickly settled centers, which occurrence is quite in contrast to such diseases as measles and scarlet fever.

Hygienic conditions, which generally bear a relationship to the incidence of an infectious disease, do not seem to play a very important rôle in the occurrence of poliomyelitis, as cases have occurred in the best hygienic surroundings.

The most striking constant about the occurrence of the disease has been its great frequency in very young children, among whom the disease is fifty times more prevalent than among young adults. This greater incidence among young children has been used as an argument favoring various modes of infection. That children use milk more generally than adults, that they wear scantier clothing, that they are less cleanly, that they are more frequently in contact with street dust, have all been used to lend support to the respective theories incriminating the milk supply, insects, lack of hygienic surroundings, or street dust as factors in the spread of poliomyelitis. None of these hypotheses is supported by epidemiologic evidence. Neither is this enormous difference explained on the basis of a greater exposure, for by analogy with other communicable diseases, such an argument cannot be supported. It can only be assumed that children are more susceptible.

The paralytic diseases which are said to occur among domestic animals simultaneously with epidemics of poliomyelitis, have neither proved to be examples of poliomyelitis, nor have they been noted in greater frequency during the prevalence of an epidemic.

While weather conditions have been suggested as supporting the hypothesis of a deficient rainfall having a direct bearing on the cause of an epidemic, experience has not confirmed the point of view. An epidemic may be declining and developing in closely approximated communities under similar meteorologic conditions.

The evidence regarding the contagiousness of the disease is very conflicting, as only a small percentage of patients give a history of contact with other infected persons. To explain the discrepancies between known contacts and instances of the disease, the existence of healthy carriers must be assumed. That such carriers exist has been proved experimentally by Flexner[5] and others.[6] The difficulties of artificially cultivating the organism which produces the disease makes impossible the routine recognition of such carriers. Recovered patients may also constitute carriers of the infectious agent.[7]

A third group, the one which in the opinion of all observers plays the most important part in the unexplainable transmission of the disease, is the group of so-called "abortive" cases, many of which pass undiagnosed. Irrespective of the type of the disease, the infectious agent, or virus is present on the mucous membranes of the nose and throat and intestines.[8] These parts of the body not only act as points of exit for the virus, but it has been shown by Flexner and his co-workers that the infection atrium is by the nose and throat. Multiplying there, the virus is readily distributed "by coughing, sneezing, kissing and by means of fingers and articles contaminated with these secretions." The virus is, moreover, resistant to high temperature and drying, so that when it is thrown off from the body, the dried secretions may, in the form of dust, constitute a potential source of infection. Regarding the conveyance of the disease by insects, "neither mosquitoes nor lice seem able to take the virus from the blood of infected monkeys." The domestic fly may conceivably act as a mechanical carrier by coming in contact with infective secretions. Flexner considers reported simultaneous instances of paralysis among domestic animals as pure coincidences, since all such instances have never proved, on proper examination, to be poliomyelitis.[9]

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5. Flexner, S.: Jour. Am. Med. Assn., 1916, **67,** 279.
6. Lucas, W. P., and Osgood, R. B.: Jour. Am. Med. Assn., 1913, **60,** 611; quoted in Jour. Am. Med. Assn., 1916, **67,** 118.
7. Flexner, S.: Am. Jour. Dis. Child., 1915, **9,** 353.
8. Sawyer, W. A.: Am. Jour. Trop. Dis. and Prev. Med., 1915, **3,** 164. See also Footnote 5.
9. Flexner, S., and Clark, P. F.: Jour. Exper. Med., 1913, **17,** 577.

The fluctuations in the severity of an epidemic, Flexner[10] explains as analogous with the fluctuation in the infectiousness of the microorganism on passage through several generations of animals, plus the variation in individual susceptibility.

Lavinder[11] notes that all epidemiologic studies relative to the disease have been directed toward either proving or disproving Wickman's hypothesis that poliomyelitis is a contact disease. While receiving the qualified approval of both epidemiologic and experimental studies, several inconsistencies make further research necessary. These inconsistencies and the fact that healthy carriers and "missed" cases constitute the greatest source of transmitting the disease, make the control of the disease very difficult.

Fronczak[12] concludes that "there must be other sources of infection than those now known." Of the cases studied in Buffalo in 1912, only 18 per cent. of the patients gave any history of contact with a previously known case. Dividing the city wards into two groups, one-half containing those with the smallest population, "it was found that where the proportion of cases was the lowest, the average density was twice as great."

Emerson[13] notes a similar occurrence in regard to the comparative prevalence in various parts of greater New York. In the Boroughs of Queens and Richmond, where the congestion was least, there were more cases in proportion to the population. Other experiences, however, emphasize the rôle played by the human carrier. The experience of the children isolated in the institutions of New York City is particularly noteworthy. In these institutions, housing a total of about 28,000 children, quarantine against visitors and new admissions was ordered when the epidemic began. Only two cases of poliomyelitis developed, "one child developing the disease on August 8, simultaneously with the child of the engineer of the institution who lived outside the grounds, his house, however, abutting the buildings of the institution, and who had been in the habit of bringing in his child in its carriage each day and keeping it in the garden near the power house." The other case was not absolutely characteristic of poliomyelitis.

An experience which bears out the assumption that hygienic conditions play no rôle is related by Emerson:

> Barren Island, situated in Jamaica Bay, is a place to which all the city garbage is brought by boat. All the city's dead animals, often two or three thousand a day, are brought here; it has no public water supply, no sewerage system, the

10. Flexner, S.; Clark, P. F., and Amoss, H. L.: Jour. Exper. Med., 1914, **19,** 195.
11. Lavinder, C. H.: Arch. Pediat., 1916, **33,** 847.
12. Fronczak, Francis E.: New York State Jour. Med., 1916, **16,** 389.
13. Emerson, Haven: Bull. Johns Hopkins Hosp., 1917, **28,** 131.

houses have no cellars, no garbage collection, and the people have few garbage cans, the household waste being thrown on the ground about the shanties. There are 17,000 people on the island, 350 of them being children, but there was no case of poliomyelitis on that island all summer. Smells, flies and insanitary conditions have prevailed, but because of its geographical position and the social condition of its people, the island is more or less isolated.

Craster,[14] reviewing the experiences of Newark in the recent epidemic, concludes that poliomyelitis is carried from place to place by human carriers. On no other basis can the spread of the disease in Newark be explained. A few instances of paralysis in lower animals which occurred during the epidemic proved not to be poliomyelitis. The occurrence of 83.8 per cent. of the cases in children under 5 years of age, and the apparent fluctuation of incidence with weather conditions, were the only outstanding features of the epidemic.

Kerley[15] studied the possibilities of contact in the spread of the disease. The instances selected were from an isolated community near his home. In all the cases detailed Kerley determined either transmission by an infected person or by an innocent carrier.

Sheppard[16] suggests that the term "contact infection" as used in regard to poliomyelitis must include instances of direct or indirect contact. The latter he refers to as the "social contact" factor. By such inclusion, any object which had previously been in intimate touch with an infected person would constitute a source of infection.

Murphy's[17] experience with poliomyelitis in the Crow Reservation upholds the contact theory. The persons affected all belonged to a group that had visited an infected district. No other individuals but those accounted for in this way developed the disease.

Richardson[18] is still of the opinion that insects may play a part in the transmission of the disease. According to his theory, rats are previously infected, the disease then being transferred to man by the agency of the flea.

Bodine[19] discusses the possible etiologic relationship of certain insects to the spread of poliomyelitis. The biennial occurrence of the disease suggests the tick as a possible carrier; the mosquito comes under suspicion by the seasonal prevalence of the disease. He refers to Rosenau's early work on the stable-fly, which in geographic distribution, its abundance under rural as well as urban conditions, correlates the incidence of poliomyelitis.

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14. Craster, C. V.: Jour. Am. Med. Assn., 1917, **68,** 1535.
15. Kerley, C. G.: Arch. Pediat., 1917, **34,** 32.
16. Sheppard, P.: New York State Jour. Med., 1916, **16,** 442.
17. Murphy, J. A.: Jour. Am. Med. Assn., 1916, **67,** 1247.
18. Richardson, M. W.: Boston Med. and Surg. Jour., 1916, **175,** 397.
19. Bodine, J. H.: Jour. Am. Med. Assn., 1916, **67,** 1872.

Dingman[20] records the occurrence of an epidemic possibly milk-borne. In three widely separated boarding houses, Dingman found several instances of poliomyelitis. All of these patients had used the milk from a particular dairy. This same dairy supplied three other houses, in which no cases occurred. On investigation, Dingman found that the residents of the last three always boiled their milk, while those of the first three used the milk raw. At the home of the dairyman there had been a case of illness in a 4-year-old boy which had been undiagnosed. The boy showed a beginning paralysis of his right leg. In the same frame dwelling, another child had been acutely ill, and at the time of observation was walking about, "dragging one foot as he walked." The cases mentioned were the only ones which had developed up to that time and for some weeks later in this locality.

The committee of the American Public Health Association concludes from a study of the disease that infection may be directly spread from person to person. They believe that the apparent inconsistencies which occur in tracing individual cases are explained by the lack of means for detecting mild cases, and by the possibility that such cases and healthy carriers are more numerous than frankly paralyzed cases. It is said:

> Many facts, such as the seasonal incidence and rural prevalence of the disease, have seemed to indicate that some insect or animal host, as yet unrecognized, may be a necessary factor in the spread of poliomyelitis, but specific evidence to this effect is lacking, and the weight of present opinion inclines to the view that poliomyelitis is exclusively a human disease and is spread by personal contact, whatever other causes may be found to contribute to its spread. In personal contact we mean to include all the usual opportunities, direct or indirect, immediate or intermediate, for the transference of body discharges from person to person, having in mind as a possibility that the infection may occur through contaminated food.

BACTERIOLOGY

In 1913, Flexner and Noguchi[21] reported the successful cultivation of a minute, filtrable micro-organism from cases of poliomyelitis. This was accomplished by inoculating tall tubes of human ascitic fluid containing sterile rabbit tissue, under anaerobic conditions, with pieces of nervous tissue obtained from poliomyelitic sources. These micro-organisms were described as minute, "globoid bodies measuring from 0.15 to 0.3 micron in diameter, and arranged in pairs, chains and masses, according to the conditions of growth and multiplication." No gas or acid was produced when various sugars and alcohols were added. The micro-organism behaved in a variable way toward the gram stain. Among its other properties were resistance to freezing,

20. Dingman, J. C.: New York State Jour. Med., 1916, **16,** 589.
21. Flexner, S., and Noguchi, H.: Jour. Exper. Med., 1913, **18,** 461.

phenolization and glycerolation.[22] Growth occurred slowly, and was best demonstrated about the sixth or seventh day. With these cultures, poliomyelitis was reproduced and the organisms again recovered from the infected animals — thus fulfilling Koch's classical requirements.

In succeeding publications on the etiologic relationship of this micro-organism to poliomyelitis, Flexner and his associates reported the cultivation of the same micro-organism from the blood of infected monkeys[23] and otherwise added much to our knowledge of the pathogenesis of this disease.[24]

It was therefore with much surprise to the students of this disease that reports were published by several independent investigators, recording the isolation of a peculiar polymorphous streptococcuslike organism from numerous cases of epidemic poliomyelitis. This organism was isolated from the tissues of the central nervous system in nine of ten cases studied by Mathers.[25] In five instances the organism was obtained after death from the cerebrospinal fluid, and in one of two cases examined, in the mesenteric lymph nodes and kidneys.

Rosenow[26] has not only cultivated the organism from the brain and cord in every instance of twelve cases of poliomyelitis, but obtained the identical organism from the tonsils in a large series of patients with this disease.

Nuzum,[27] working independently of these investigators, contributes similar results. Later, Nuzum[28] also reported successful growth of the identical organism from the cerebrospinal fluid in forty of fifty fluids examined.

All of these investigators were, moreover, successful in reproducing the disease by injection of their cultures into rabbits and monkeys. Rosenow and his co-workers report successful results with guinea-pigs, cats and dogs; Nuzum and Herzog with lambs, all of which animals have heretofore been regarded as insusceptible to the disease.

The micro-organism obtained by these various investigators resembles in several respects the globoid bodies described by Flexner and Noguchi. Thus, it is resistant to glycerolation, passes through a Berkefeld filter and is neurotropic. Morphologically, a tendency to extreme irregularities was noted.

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22. Flexner, S., and Amoss, H. L.: Jour. Exper. Med., 1917, **25,** 539.
23. Amoss, H. L.: Jour. Exper. Med., 1914, **19,** 212.
24. Flexner, S., et al.: Jour. Exper. Med., 1913-1917, **18-25,** inclusive.
25. Mathers, George: Jour. Am. Med. Assn., 1916, **67,** 1019; Jour. Infect. Dis., 1917, **20,** 113.
26. Rosenow, E. C.; Towne, E. B., and Wheeler, G. W.: Jour. Am. Med. Assn., 1916, **67,** 1202.
27. Nuzum, J. W., and Herzog, M.: Jour. Am. Med. Assn., 1916, **67,** 1205.
28. Nuzum, J. W.: Jour. Am. Med. Assn., 1916, **67,** 1437

Depending on variations in artificial cultivation, the micro-organism appears to grow large or small. "The larger forms," according to Rosenow, "tend to grow smaller, so that frequently at the end of twelve or fourteen days, nothing but the tiny globoid bodies, single, in pairs, chains and clumps could be found in the same tubes. After about three weeks, the organism became both larger and smaller; the small forms get beyond the limit of visibility, and nothing but rather large oval cocci staining a pinkish tint with the Giemsa stain are seen." The globoid bodies, Rosenow concludes, are the form which this polymorphous streptococcus assumes under anaerobic conditions.

Rosenow and Towne,[29] in a more recent study, assert that the globoid bodies which Flexner and his associates have considered to be the cause of poliomyelitis are produced by fission of the diplococcus recently isolated.

Rosenow, Towne and Wheeler[30] record an experiment indicating the protection of a monkey against ordinary poliomyelitic virus by the injection of their organism (under conditions which apparently exclude the possibility of any of the globoid bodies being present).

The Rockefeller workers are unwilling to concede that the micro-organism described by these various investigators bears any causal relationship to poliomyelitis.[31]

Amoss,[32] in a renewed study of the globoid bodies, contends that "while a variety of ordinary bacteria has been obtained by various workers, the globoid bodies represent a peculiar species, and thus far alone have sufficed to produce typical poliomyelitis in monkeys by intracerebral inoculation."

Bull[33] insists that the organism described by these various investigators is a streptococcus and that the lesions experimentally produced by its injection are in no way identical with those seen in poliomyelitis. These conclusions he reaches from an experimental study of the inoculation of several species of laboratory animals with cultures of ordinary streptococci and with cultures of streptococci obtained from poliomyelitic sources. Some of the animals developed meningitis with metastatic lesions elsewhere, referable to the streptococcus. "No distinction in the character or frequency of the lesions could be determined between the streptococci derived from poliomyelitic patients and from other sources."

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29. Rosenow, E. C., and Towne, E. B.: Jour. Med. Research, 1917, **36,** 175.
30. Rosenow, E. C.; Towne, E. B., and Wheeler, G. W.: Jour. Am. Med. Assn., 1917, **68,** 280.
31. Editorial: Jour. Am. Med. Assn., 1917, **68,** 1122.
32. Amoss, H. L.: Jour. Exper. Med., 1917, **25,** 545.
33. Bull, C. G.: Jour. Exper. Med., 1917, **25,** 557.

Milton J. Rosenau[34] succeeded in transferring the poliomyelitic virus (using ordinary virus) from rabbit to rabbit through eight successive generations. He obtained positive results in twenty-two out of fifty-four inoculated in various ways. He found that young rabbits were more susceptible to the infection than older ones. Rosenau observes that "the lesions while definite and consistent throughout the series, lack the distinctive features of the pathologic picture of poliomyelitis in man and the monkey."

Gauss[35] confirms Nuzum's findings of a micro-organism in the cerebrospinal fluid of patients with poliomyelitis. As controls, Gauss cultured the cerebrospinal fluid obtained from fifty patients having "scarlet fever and associated exanthems," but in none of these did he obtain the organism mentioned.

Abramson[36] states that in the recent epidemic in New York, the Health Department laboratories there "cultured upward of 1,200 fluids from patients with acute poliomyelitis in all stages of the disease. The medium used was 1 per cent. glucose ascites agar under aerobic conditions. Except for a few evident contaminations, cultures remained sterile."

Kolmer[37] cultured various tissues obtained from cases of poliomyelitis. He isolated four varieties of bacteria, a streptococcus, a diplococcus, diptheroids and gram-negative bacilli. He was unable to produce paralysis by animal inoculation with the organisms isolated.

From specimens of brain and cord tissue taken from poliomyelitic sources, Greeley[38] cultivated a pleomorphic organism which he was able to transmute from a streptococcuslike form to a bipolar bacillus. Cats and rabbits were intravenously inoculated, the results varying from "symptoms analogous to those described as cat distemper by the veterinarians" to degrees of paralysis of various muscles.

Hektoen[39] summarizes the status of the recent findings in regard to their bearing on the etiology of poliomyelitis as follows:

> The micrococcus recently found in poliomyelitis and in poliomyelitis monkey virus appears to have many properties in common with the virus of the disease and with the minute organism described by Flexner and Noguchi. Under the conditions in which this minute organism was grown, the coccus appears to grow much in the same way and to assume very minute forms; the coccus readily passes the filters commonly used in the study of poliomyelitis virus, but the very important question, whether it will pass the finest filters that are said to let the active agent in the virus go through, has not been detetrmined; like the

34. Rosenau, Milton J., and Havens, L. C.: Jour. Exper. Med., 1916, **23,** 461.
35. Gauss, H.: Jour. Am. Med. Assn., 1917, **68,** 779.
36. Abramson, H. L.: Jour. Am. Med. Assn., 1917, **68,** 546.
37. Kolmer, J. A.; Brown, C. P., and Freese, A. M.: Jour. Exper. Med., 1917, **25,** 789.
38. Greeley, Horace: Med. Record, New York, 1917, **91,** 56.
39. Hektoen, L.: Boston Med. and Surg. Jour., 1917, **176,** 687.

virus the coccus under certain conditions is strongly resistant to the prolonged action of glycerol; in monkeys, injections of the coccus three to four culture generations removed from the primary culture have produced conditions absolutely indistinguishable, clinically and anatomically, from the classical induced poliomyelitis in this animal, but the possibility that what may prove to be the true agent of poliomyelitis may be carried along with the coccus under such conditions, must be considered; in young rabbits the coccus causes a flaccid paralysis and also other nervous symptoms, as well as lesions that correspond almost completely to lesions regarded as distinctive of poliomyelitis; but the crucial confirmation test of production of poliomyelitis in monkeys by inoculation of cultures or other materials from such rabbits is still largely lacking; like the active agent in poliomyelitis virus, the coccus, too, is strongly neurotrophic, and in the human disease, as well as in the inoculated rabbit and monkey, it locates with what seems a special preference in the central nervous tissues, in microscopic preparations of which it is demonstrable without great difficulty and from which it can be obtained in culture.

To conclude: The exact significance of this coccus in epidemic poliomyelitis cannot be determined now. The number of cases studied for its presence is too small to permit the conclusion that it occurs constantly in the disease or any form of the disease; in the few instances in which injections of cultures have resulted in a condition indistinguishable from what is accepted as poliomyelitis in the monkey, the possibility that another and more important microbe may have been present cannot be excluded; the true poliomyelitic nature of the very interesting lesion caused by the coccus in rabbits has not been confirmed by proper tests on monkeys; and we lack also the results of extended immunization experiments. In any event, a most interesting coccus has been found that merits study for its own sake as well as on account of the close relation its brief history bears to poliomyelitis.

SYMPTOMATOLOGY

According to Flexner,[5] the incubation period of poliomyelitis "is subject to wide limits of fluctuation. In certain instances it has been as short as two days; in others it has been two weeks or possibly even longer. But the usual period does not exceed about eight days."

Neustaedter[40] speaks of a case in which an incubation period of one day was established. "It was in a physician's child and the father brought the infection after staying in the house of a relative with a poliomyelitis patient for thirty-six hours. Twenty-four hours after he had arrived home, his child was stricken with fever, which lasted two days. On the third day, the child's lower extremities became paralyzed."

While Wickman's classification of eight types of poliomyelitis has generally formed the basis of the presentation of the symptomatology of poliomyelitis, the classification of Peabody, Draper and Dochez is much simpler.[41]

These are:

1. The abortive group.
2. The cerebral group.
3. The bulbospinal group.

40. Neustaedter, M. N.: New York Med. Jour., 1916, **104,** 145.
41. Bulletin, Dept. Health, City of New York, 1916, **5,** 217.

Ruhräh[42] further subdivides the abortive group into five classes, four of which are given by Wickman, as follows:

1. Those with a course of a general infection.
2. Those showing meningeal irritation.
3. Those with marked pains suggesting an influenza.
4. Those with gastro-intestinal disturbances.

To these he adds,

5. The anginal form, those beginning with a sore throat.

The classification suggests the extremely varied manifestations of this disease. Many observers do not favor the use of the term "abortive"; epidemiologists, on the ground that the term minimizes the equal importance of this type in the transmission of the disease; clinicians, that poliomyelitis should in any of its forms be recognized as an acute infectious disease, which may or may not be associated with paralysis. Emerson suggests the terms preparalytic, nonparalytic and paralytic.

Draper[43] makes a very interesting grouping of the disease, according to its clinical course:

> The first group, called the dromedary group, shows the curious phenomenon of two different periods of illness with an interval of wellbeing. In the second group this period of comparative wellbeing is not present, but there is a sustained period of indisposition of varying intensity. This is called the straggling group. In the third group only do we have all signs pointing from the start to meningeal and nervous involvement. This we called the sudden onset group. A striking thing is that the second portion of the first two groups is very similar to the whole course of the sudden onset group.
>
> In the first two groups there develops a picture of general systemic infection from which the child appears to recover completely, or in part, and then to receive a second blow directly on the cerebrospinal tract. Because of the two distinct groups or humps of symptoms, the analogy to the arrangement of the dromedary's back was taken to express the type figuratively. The temperature curve often shows two elevations, but the figure refers to all the signs and symptoms of each group or hump, whether or not there is a rise of temperature in both. The total duration of the combined phases may vary from a few hours to many days, while the intensity of either phase, the systemic or the meningeal, may touch the extreme of violence or such mildness as to escape detection. In the systemic stage, or first hump, of a well-marked case the clinical picture is that of almost all of the acute infections of childhood—a flushed, uncomfortable, feverish child; in the meningeal stage, or second hump, there are added the special signs and symptoms of meningeal irritability. It becomes at once obvious that the so-called "abortive" types are in all probability examples of the disease in which the first stage or hump constitutes the entire course of the malady, the meningeal stage either never occurring, or, if it does, in so slight a form as to pass unrecognized. The term "abortive" consequently gives a wrong impression, for the termination of the disease at the end of the systemic phase is the more usual occurrence. It would be more correct to speak of acute poliomyelitis, with or without paralysis.

42. Ruhräh, J.: Am. Jour. Med. Sc., 1917, **153,** 178.
43. Draper, G.: Jour. Am. Med. Assn., 1917, **68,** 1153.

Draper interprets these periods in the cycle of poliomyelitis infection as analogous with the primary lodgment of the virus in the spleen and bone marrow and its subsequent entrance into the cerebrospinal spaces, as illustrated experimentally in monkeys by Flexner and Amoss.[44] The first febrile period of the dromedary or straggling type would correspond to the first part of this cycle, the second hump of the dromedary type to the fixation of the virus in the central nervous system. Draper further says:

> Experiment has shown that the virus gains entrance with difficulty to the central nervous system tissues of monkeys subjected to intravenous inoculation. In human subjects the number of cases recognized as never giving the slightest indication of central nervous system invasion is steadily growing with our experience.
>
> In families in which a frank paralysis case occurs, there are found so frequently other children who present persistently the same symptoms which the paralyzed child has shown in the preliminary hump, that one is led to suppose that these may be examples of infection in which no penetration of the meninges occurred.
>
> In the straggling group the same interpretation applies, the only difference being that the individuals present a continuous course with a fusion of the symptoms of the two stages. Sudden onset cases may be looked on as examples of the malady in which the systemic stage has been overlooked because of its triviality. In this group the spectacular and violent picture of meningeal disease dominates and drives the memory of the slight preceding indisposition from the mother's mind. It is difficult under these circumstances to get from the family any history of the child being unwell during the preceding days.

All observers note the occurrence of fever as the most constant initial symptom. Fischer[45] states that the fever usually persists from three to five days, though it may last from seven to fourteen days. In severe forms, a sudden rise occurs, lasts about forty-eight hours and declines by crisis. These is no characteristic febrile curve that is pathognomonic of poliomyelitis.

Neustaedter[40] considers "the paleness of nasopharyngeal mucosa and its edema, accompanied in the early stage by a serous and frothy transudate, as constant and pathognomonic of the earliest prodromal stage."

Colliver[46] gives a table of the early symptoms. He mentions:

1. Changes in disposition.
2. Fever and anorexia, with constipation or diarrhea.
3. Lack of coordination.
4. Hypersensitiveness of skin.
4. Drowsiness.
6. Pain.

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44. Flexner, S., and Amoss, H. L.: Jour. Exper. Med., 1914, **20,** 249.
45. Fischer, L.: Med. Rec., New York, 1917, **91,** 52.
46. Colliver, J. A.: Arch. Pediat., 1917, **34,** 263.

7. Tremor of certain groups of muscles.
8. Hyperacusis in the bulbar cases.

Koplik[47] reviews the clinical histories of the representative types of the disease. He describes the so-called abortive type. This type can be recognized so as to leave no doubt as to its distinct identity.

A child of 5 years is attacked with a headache, slight malaise and an attack of vomiting lasting five days, intense pain in both lower extremities radiating to the soles of the feet and worse at night, slight pain in the nape of the neck, lassitude, cerebellar gait on walking, increased reflexes in the lower extremities, and rectal temperature above 100.5 F. In ten days the pains have disappeared the child is well and wants to go out and play. The abortive cases present prodromes such as headache, weakness, diminished reflexes and pain in the nape of the neck with or without vomiting and fever; still do not present paralysis, and the patient recovers.

The spinal or bulbospinal type is the most common, and gives the disease its name. Its symptoms are now well known and well defined.

Draper considers the "spine sign" as one of great value in early diagnosis. This consists in an attempt to flex the spine forward. Any such manipulation produces pain and is resisted by the patient.

Fischer[48] considers Colliver's sign as one of the valuable preparalytic evidences of the disease. This is "a peculiar twitching, tremulous or convulsive movement. It usually affects a part or whole of one or more limbs, the face or jaw. It may also affect the whole body."

Gordon[49] calls particular attention to the examination of the reflexes in the early diagnosis of suspicious cases. The patellar and achilles reflexes should be investigated. Even a diminution of these reflexes should put one on guard for this disease, particularly during an epidemic.

Sheffield[50] notes the frequent onset of poliomyelitis with sore throat, "the tonsils presenting either simple congestion or also small greyish-white deposits." When such symptoms are associated with a palatal paralysis, the picture of diphtheria may be closely simulated. The early symptoms of the disease may suggest laryngeal diphtheria,[51] appendicitis,[52] bronchopneumonia,[53] or may duplicate any meningeal disease. While all observers agree that many cases occur which remain undiagnosed, during an epidemic the diagnosis is apt to be made with-

47. Koplik, H.: Jour. Am. Med. Assn., 1916, **67,** 310.
48. Fischer, L.: Med. Rec., New York, 1916, **90,** 194.
49. Gordon, A.: New York Med. Jour., 1916, **104,** 583.
50. Sheffield, H. B.: Med. Rec., New York, 1916, **90,** 330.
51. Polozker, I. L.: Jour. Michigan Med. Soc., 1917, **16,** 100.
52. Le Boutillier, T.: Am. Jour. Med. Sc., 1917, **153,** 188.
53. Louria, L.: Arch. Pediat., 1916, **33,** 853.

out justification. Thus the Harvard Infantile Paralysis Commission[54] report that of the 187 calls received, only 123 proved to be poliomyelitis. The remaining sixty-four instances embraced many diseases (infectious diarrhea, gastro-enteritis, influenza, vaccination, hysteria, tuberculosis, "mild digestive upsets," "nervousness"). Among this group there were also four cases of meningitis — two of the epidemic type, one tuberculous and one luetic.

Wilson[55] classifies the prominent prodromal symptoms, based on a study of 400 histories of patients with poliomyelitis admitted to the Willard Parker Hospital. Fever was present as an initial symptom in 334 cases. Only 2 per cent., on careful investigation, gave no history of fever. Vomiting was an initial symptom in sixty-seven cases, as an early symptom in 132 cases. "One hundred and fifty-six cases gave a definite history of persistent constipation for two or more days, resisting ordinary catharsis." Persistent drowsiness was noted in 72 per cent. of the cases. Other nervous symptoms observed were irritability, hyperesthesia, tremor, delirium, convulsions. Profuse sweating was noted in forty-five cases. This was usually out of proportion to the fever present. Wilson considers a careful history as suggestive, particularly during an epidemic.

Neal and Dubois[56] record two cases of poliomyelitis associated with blindness. In one instance, sight was restored at the end of about three months. The second instance was a more recent one in which the blindness was still present at the time of their report.

No characteristic eye signs have occurred in poliomyelitis, according to Hansell.[57] The eye complications varied considerably as one would expect following such a generalized cerebrospinal infection.

It seems to be generally agreed that the examination of the cerebrospinal fluid in poliomyelitis offers the most useful single procedure available in the early diagnosis of the disease.

Abramson[58] mentions the usual findings as follows:

1. Fluid is increased and under pressure.
2. Fluid is clear as a rule, but may be opalescent.
3. Albumin and globulin increased.
4. Fehling's solution reduced in the majority of instances.
5. Cells increased.
6. The predominating cell type is the small, mononuclear, which may constitute 90 per cent. of the cells present. In opalescent fluids

54. Report Harvard Infantile Paralysis Commission, Boston Med. and Surg. Jour., 1917, **176,** 637.
55. Wilson, May G.: Am. Jour. Dis. Child., 1917, **13,** 506.
56. Neal, J. B., and Dubois, P. L.: Am. Jour. Med. Sc., 1916, **152,** 313.
57. Hansell, H. F.: Am. Jour. Dis. Child., 1917, **13,** 516.
58. Abramson, H. L.: Med. Rec., New York, 1916, **90,** 793.

one may find the polynuclears increased, as high as 75 per cent. Abramson has also noted the presence of glialike cells and endothelioid cells.

Zingher[59] thinks the cerebrospinal fluid quite characteristic even in its early stages. He says:

> There is an increase in the total number of cells, from twenty to 600 or more. These cells are mostly lymphocytes, with a small proportion of large mononuclear and polynuclear cells. During the earliest stages of the disease there may be a transient relative and absolute polynucleosis. There is an increased amount of albumin, globulin and substance which reduces Fehling's solution. Often on the surface of the spinal fluid, as it is being drawn into a test tube, a thin filament of fibrin appears, which breaks up into small flakes when the fluid is gently shaken.

Zingher describes what he terms a ground-glass appearance when such fluids are viewed by transmitted light. The suggestion of ground glass is due to the increased number of cells in the fluid. This appearance is not present in a fluid that has been standing several hours. The same author speaks of a so-called "foam test." All spinal fluids when shaken in a test tube create a foam, but that formed by shaking a test tube half filled with spinal fluid from a poliomyelitic patient is much denser and more persistent. Zingher warns against deductions with a spinal fluid that is not absolutely free from blood.

Josephine B. Neal[60] discusses the laboratory aids in the diagnosis of poliomyelitis. Most evidence furnished by such procedures as are available are of a corroborative nature and not absolutely diagnostic. The neutralization test is too complicated. In this test, a mixture of a fatal dose of an active virus with the suspected fluid, obtained during the stage of recovery, is incubated and injected intracerebrally into a monkey. Failure of the disease to develop indicates neutralization of the virus. The examination of the cerebrospinal fluid helps differentiate from the early stages of meningitis and from meningismus. Large monoculear cells, which may have some diagnostic significance, are occasionally noted. "Two rare types of spinal fluids sometimes occur in poliomyelitis when the hemorrhagic process has been more than usually extensive. The first of these is of true hemorrhagic character, the red blood cells being evenly diffused throughout the fluid. When collected in successive tubes, the specimens are all homogeneous, showing no change in the intensity of the hemorrhage. This serves to differentiate it from bloody fluids obtained by the accidental puncture of a vein. The second of these rarer fluids illustrates the so-called syndrome of Froin. It has a characteristic yellow color and coagulates

59. Zingher, A.: Jour. Am. Med. Assn., 1917, **68,** 817.
60. Neal, Josephine B.: New York Med. Jour., 1916, **104,** 167.

spontaneously." These fluids mentioned are not pathognomonic of poliomyelitis.

In the diagnosis of poliomyelitis, Dubois[61] considers the laboratory more of value "by what it rules out." The important features are the increased cell count, which may be "marked, moderate or slight," the positive globulin and the reduction of Fehling's solution. Frequently cells are so degenerated as to make differential classification difficult.

Among the examinations of cerebrospinal fluid made by the Harvard Paralysis Commission[54] there were thirty-one instances of fluids from patients who were ultimately proven not to have poliomyelitis. In two instances the cell count was 12 and 15, respectively. In all other instances the cell count was below 10. In the positive cases, the count ranged from 34 to 1,980 per cubic millimeter.

Felton and Maxcy[62] find the colloidal gold reaction an aid in the laboratory diagnosis of poliomyelitis. The fluids were taken from patients in various stages of the disease. "The gold chlorid curve given in these cases is singularly constant and occurs in the so-called luetic zone." After the subsidence of the acute illness the reaction may be most prominent in the "meningitic zone."

Jeans and Johnson[63] also applied the gold-chlorid test, with very similar results, the reaction occurring in the lower dilutions. Even when a reaction does take place in the middle or meningitic zone, it will not be restricted to this particular zone, but also embrace the first zone.

Hoyne and Cepelka[64] record the leukocyte counts in thirty patients with poliomyelitis. Leukocytosis was the rule and usually persisted for at least two weeks.

PROGNOSIS

Prognosis is a hazardous venture, according to Abramson.[58] Cases which appear grave may clear in twenty-four to forty-eight hours and present the most astonishing improvement. There are others which appear mild, develop fulminating symptoms and the patients die in a few hours. In adults, the disease seems more violent. Relapse occasionally occurs after a period of apparent improvement.

In Weisenburg's experience,[65] "the mildest cases often developed the severest infection, while a very sick child might have only limited paralysis." The most fatal cases in his experience were those of the so-called Landry type.

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61. Du Bois, Phoebe: Arch. Pediat., 1916, **33,** 856.
62. Felton, L. D., and Maxcy, K. F.: Jour. Am. Med. Assn., 1917, **68,** 752.
63. Jeans, P. C., and Johnson, M. R.: Am. Jour. Dis. Child., 1917, **13,** 239.
64. Hoyne, A. L., and Cepelka, F. P.: Jour. Am. Med. Assn., 1916, **67,** 666.
65. Weisenburg, T. H.: Jour. Am. Med. Assn., 1916, **67,** 1872.

La Fetra[66] warns that one should be particularly cautious in giving an absolutely good prognosis in cases of facial paralysis.

Ruhräh[42] says that "the severity of the initial symptoms bear no relation whatever to the course of the disease, as one sees a very mild onset followed by most extensive paralysis and even death, and other cases coming in a most fulminating manner which subsequently clear up entirely."

The case mortality in this epidemic was much higher than that of previous ones. In New York City, it reached 26.96 per cent. In New York State, not including the city, it was 21 per cent.[2] The younger the child, the higher the mortality rate.

Partial statistics available at present give variable figures as to the percentage that escape paralysis. The general impression exists that there are a large number of such instances, which probably remain undiagnosed. In one period of six weeks during the New York epidemic, in an analysis of 2,053 cases of patients discharged from the hospitals under the control of the New York Health Department,[67] it was stated that 66 per cent. showed some paralysis; 15 per cent. had never had paralysis, and 15 per cent. showed a disappearance of previously existing paralysis. The last figure will exhibit a proportionate increase with the length of intervals following the acute infection.

Lovett and Martin[68] have devised a spring balance test by which the efficiency of any muscle or group of muscles may be measured and expressed in percentages. With the patient in a certain standard position, the test is performed by having the patient hold a position against the pull of a spring balance. The resistance exerted represents the muscular strength. By the use of this test these observers have determined that the distribution of the paralysis is much more general than has been ordinarily supposed; that cases of so-called abortive paralysis are often cases in which the paralysis is of too mild a degree to be detected by the usual methods of examination; that the proportion of partially paralyzed to totally paralyzed muscles is about 9 to 1. They also demonstrated that spontaneous improvement occurs for a much longer period and to a greater degree than has generally been thought possible.

Sayre[69] and others doubt the significance of the reaction of degeneration, since despite the presence of the reaction of degeneration, muscles may regenerate.

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66. La Fétra, L. E.: Arch. Pediat., 1916, **33,** 865.
67. Medical News: Jour. Am. Med. Assn., 1916, **67,** 1236.
68. Lovett, R. W., and Martin, E. G.: Jour. Am. Med. Assn., 1916, **66,** 729. Lovett, R. W., and Martin, E. G.: Am. Jour. Orthop. Surg., 1916, **14,** 415.
69. Sayre, R. H.: New York Med. Jour., 1916, **104,** 1029.

TREATMENT

Inasmuch as the epidemiologic knowledge of this disease is incomplete, the prophylactic measures which are advised are such as apply to other communicable diseases.

"Of first importance," says Emerson, "is the more general recognition by practitioners of nonparalytic cases through clinical observation and laboratory procedures."

Emerson offers an outline of necessary administrative measures for the control of the disease as follows:

1. The requirement that all recognized and suspected cases be promptly reported.

2. Isolation of patients in screened premises. The duration of infectivity being unknown, the period of isolation must necessarily be arbitrary. Six weeks has been recommended by the Conference of State and Territorial Health Officers with the Surgeon-General of the Public Health Service as sufficient, and this period has been generally accepted throughout the United States.

3. Disinfection of all body discharges.

4. Restriction of the movements of intimate associates of the patient, so far as practicable. This should include at least exclusion of the children of the family from schools and other gatherings.

5. Protection of children, so far as possible, from contact with other children or with the general public during epidemics.

6. Observations of contacts for two weeks after the last exposure.

Interstate and intrastate quarantine regulations which at the height of the 1916 epidemic in some instances bordered on the hysterical, have now been placed on a uniform basis.[70] In August, 1916, a special conference of state and territorial health officers met with the United States Public Health Service for the consideration of measures for prevention of the spread of poliomyelitis. The transactions of this conference are available in a Public Health Service publication.[71]

Since the nasopharynx is generally believed to be the point of entry for the virus of poliomyelitis, Bryant[72] recommends measures for the routine treatment of the nasopharynx. The correction of any nasal disturbance and the use of various antiseptic and astringent solutions forms the basis of the treatment. Hydrogen peroxid, silver salts and the salts of iron are among the local remedies advised.

Whittemore[73] suggests the insufflation of the nose and throat with kaolin as advised for diphtheria carriers by Hektoen and Rappaport.

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70. U. S. Public Health Reports, Reprint 361.
71. U. S. Public Health Bull. 83.
72. Bryant, W. S.: New York Med. Jour., 1916, **104,** 727.
73. Whittemore, W. S.: Boston Med. and Surg. Jour., 1916, **175,** 231.

Roper[74] discusses the advisability of tonsillectomy, since Rosenow claims that the tonsil may harbor the offending organism.

Emerson voices the opinion of many others in some remarks regarding treatment. He says:

> There is no specific treatment of established value in poliomyelitis. During the persistence of the acute symptoms of the disease, the important principles of treatment are rest in bed, symptomatic relief and passive support for the prevention of deformities. Active measures during this stage are not only useless, but are apt to cause serious and often permanent injury. Hospitalization of patients where possible is to be encouraged. The best chances of recovery from residual paralysis demand skilful after-care, often long continued, and always under the direction of a physician familiar with the neurologic and orthopedic principles of treatment. The provision of such after-care often becomes a community problem, demanding the cooperation of all available agencies, social and professional.

While lumbar puncture affords the best single therapeutic agent, some cases showed good recoveries without any other than expectant treatment. Many think that the good results reported by the use of various intraspinal measures are in the main attributable to the lumbar puncture per se.

SERUM THERAPY

Flexner and Lewis have shown that monkeys that recover from poliomyelitis are immune to subsequent inoculation with the virus of the disease. This was later shown to be due to the presence of neutralizing substances in the blood of such animals. It was also demonstrated that the blood serum of an individual that has recovered from poliomyelitis possesses similar neutralizing principles. By utilizing immune serums, Flexner and his co-workers were able to prevent the occurrence of paralysis in infected monkeys. The application of this measure in the treatment of epidemic poliomyelitis in man was first made by Netter.[75] He employed the serum of individuals who had had the disease at some previous time—even as long as thirty years previously.

Flexner[76] discusses the application of this method. He says:

> The dose of the serum, which must, of course, be sterile, but need not be inactivated, should be determined by the age of the patient, and will, in part, be determined by the quantity of serum available. Probably doses ranging from 5 to 20 c.c. will be found suitable, the injection to be repeated several times at twenty-four-hour intervals according to clinical conditions and indications. The effects of the immune serum should be sought in the checking of the progress of the disease, namely, the prevention or minimization of the paralysis when employed in the preparalytic stages, and the arrest of its extension when used in progressing paralytic conditions. Since the immunity sub-

74. Roper, J. C.: Med. Rec., New York, 1916, **91,** 1139.
75. Netter, A.: Bull. de l'Acad. de méd., Paris, 1915, **74,** 403.
76. Flexner, S.: Jour. Am. Med. Assn., 1916, **67,** 583.

stances have been determined by neutralization tests to persist in the blood for many years, it is probable, as Netter had indicated, that persons who have passed through an attack of poliomyelitis many years earlier may be utilized as sources of the serum; reasoning from analogy, it would probably be advantageous to prefer persons whose attack was less remote so as to insure as high concentration of the immunity bodies as possible. The conditions surrounding the injection of the serum into the meninges are identical with those observed in the analogous case of epidemic meningitis. Before each dose of serum is injected a suitable quantity of the cerebrospinal fluid is to be withdrawn, and the injections should be made slowly. In choosing the person who is to serve as the source of blood from which the immune serum is to be derived, precaution should of course be taken to secure a healthy donor; it would be advisable to fortify the usual clinical examination by a Wassermann test.

Zingher[59] describes the methods of obtaining and preparing serum from immune donors. He receives the blood in small square bottles in quantities of 1 to 2 ounces; the bottles are slanted to allow larger surface for clotting and separation of serum. The serum is centrifuged, and may then be preserved by the addition of 0.2 per cent. trikresol solution. (This may increase the irritant effect of the serum.) After standing in the icebox for forty-eight hours, a sediment which forms on the addition of trikresol is removed and the serum is passed through a Berkefeld filter and bottled. When these facilities are not obtainable, the serum may be defibrinated and centrifuged, or the serum separated in the usual way and used immediately. No inactivation of serum is necessary.

Zingher tabulates the results from the use of serums obtained from individuals who had had the disease at intervals varying from a couple of months to over thirty years previously. He did not observe any difference in the results obtained. Indeed, Zingher considers it problematic whether such results as are obtained may not be due to the cellular response created by the intraspinal injection of a protein, and therefore not a specific one. Of fifty-four preparalytic cases treated with immune serum, forty-four patients remained free from paralysis, and of the remaining ten, five made a complete recovery. Of ten patients treated with normal serum, nine remained free from paralysis and one died. Among the paralytic cases the mortality was high despite the use of serum, which Zingher explains on the ground of the extreme severity of these cases. There were forty-five deaths in a total of 119 patients treated.

Schwarz[77] treated twenty-one patients with serum and a similar number by the expectant plan. Of the former, nine recovered without paralysis. Of the latter, seventeen recovered without paralysis.

Petty[78] reports eleven patients treated by subdural administration of immune serum. Striking results were not obtained.

77. Schwarz, H.: Arch. Pediat., 1916, **33,** 859.
78. Petty, O. H.: New York Med. Jour., 1916, **104,** 1190.

In a series of patients treated by the Harvard Infantile Paralysis Commission, there was no evidence of paralysis in 69 per cent. of the total number seen in the preparalytic stage. In a series of eighty-five cases furnished them by Draper, in which no serum was used, 56 per cent. developed no paralysis. The difference is not sufficiently striking to warrant any definite conclusions.

Sophian[79] was unable to note any greater advantages from the use of immune serum than from normal horse serum.

Wells[80] thinks that the best results are obtained by combined intravenous and intraspinal injections. The intravenous route has the advantage of the utilization of a much greater amount of serum, and, according to Wells, is a logical procedure, because the peculiar distribution of poliomyelitic lesions suggest a hematogenous infection.

Draper[43] discusses the apparent discrepancies between statistical proofs of the use of immune serum and the clinical impressions received. On the basis of the analogy drawn by Draper between the two "humps" of the clinical picture and the interception of the infecting virus by the spleen and meninges, the use of any serum, homologous or otherwise, should theoretically await the onset of meningeal symptoms. Since the clinical recognition of this onset is not always a simple matter, the delay in the use of serum may mean the loss of valuable time. Since Flexner and his associates have shown experimentally that the injection of serum intraspinally renders an animal more susceptible to an injection of poliomyelitic virus, the entire procedure may be a dangerous one. In the final decision, each case must be considered by itself.

Amoss and Chesney consider the use of serum a procedure of definite therapeutic value. When given under suitable conditions, its administration is harmless. They recommend that it be used during the first thirty hours of the disease, and advise the administration of large doses, utilizing the intravenous and intraspinal routes. According to their studies, the serum is more efficacious in preventing paralysis than in effecting its retrogression.

Flexner and Amoss[81] present an interesting study explaining the alleged beneficial results derived from the intraspinal injection of normal serum in poliomyelitis. They demonstrate that the injection of any serum increases the permeability of the meninges. This would allow the passage of any neutralizing principles from the blood to the cerebrospinal fluid. Since in the very early stages of the disease, immunity principles do not exist in detectable amounts in the blood,

79. Sophian, A.: Jour. Am. Med. Assn., 1916, **67,** 427.
80. Wells, C. W.: Jour. Am. Med. Assn., 1916, **67,** 1211.
81. Flexner, S., and Amoss, H. L.: Jour. Exper. Med., 1917, **25,** 499.

the rationale of the use of normal serum comes into question. On the same basis the use of immune serum is an obviously rational procedure, and may prove curative.

Rueck[82] reports three cases of poliomyelitis treated successfully by transfusion of the citrated normal blood of adults.

Duncan[83] reports the use of the patient's own spinal fluid for intramuscular injection. He removed 10 c.c. by lumbar puncture and injected 1 c.c., and later 0.5 c.c. more, intramuscularly. The patient recovered.

Meltzer[84] objects to the method as irrational and dangerous.

Ager[85] replies to Meltzer's criticism. He says that in the use of the method as supervised by him, 60 to 70 c.c. of spinal fluid were removed and only 0.5 to 3 c.c. reinjected. There were no bad results. Some were strikingly improved. Ager, however, is skeptical about its ultimate value or that of any present method of treatment. He believes that the only method of value will be the production of a concentrated serum from an immunized lower animal.

Neustaedter and Banzhaf[86] report some neutralization experiments obtained by the use of an antipoliomyelitis horse serum. Since previous investigators had not been successful, he attempted to immunize horses by injections of the filtrates of brain and cord (from human poliomyelitic sources) which had previously been incubated with a proteolytic ferment. This was done in order to obtain a possible endotoxin. The neutralization experiments were positive without exception.

Nuzum[87] reports the production of a poliomyelitic antiserum. He immunized sheep by successive injections of the organisms he previously described as the causative agent in the disease. The antiserum prepared contained agglutinins, opsonins and complement fixation bodies as well as antibactericidal properties. Its therapeutic possibilities have not been ascertained.

Mathers and Tunnicliff[88] report the occurrence of specific opsonin in the blood of convalescent poliomyelitis patients. In conjunction with Dr. Katherine Howell, Tunnicliff has found that "the serum of rabbits immunized against different strains of poliomyelitic cocci contains antibodies in high concentration, which are apparently specific for the poliomyelitic organisms."

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82. Rueck, G. A.: Med. Rec., New York, 1916, **90,** 587.
83. Duncan, C. H.: New York Med. Jour., 1916, **104,** 342.
84. Meltzer, S. J.: New York Med. Jour., 1916, **104,** 477.
85. Ager, L. C.: New York Med. Jour., 1916, **104,** 573.
86. Neustaedter, M., and Banzhaf, E. J.: Jour. Am. Med. Assn., 1917, **68,** 1351.
87. Nuzum, J. W.: Jour. Am. Med. Assn., 1917, **68,** 24.
88. Mathers, G., and Tunnicliff, R.: Jour. Am. Med. Assn., 1916, **67,** 1935.

Meltzer[89] offers an interesting discussion on the treatment of poliomyelitis by the intraspinal injection of epinephrin chlorid and the maintenance of artificial respiration by intrapharyngeal insufflation. Meltzer quotes Peabody, Draper and Dochez as declaring that, "it is often extremely difficult to reconcile the clinical symptoms which are referable to pontine lesions with the actual necropsy findings." Such symptoms, Meltzer assumes, may be due to local edemas which disappear as soon as the circulation stops. Since epinephrin exerts a favorable influence on local edemas, its use in poliomyelitis suggested itself. Meltzer offers the further hypothesis that epinephrin may be specifically antagonistic to the virus of poliomyelitis. The Rockefeller workers have shown that the abdominal sympathetic ganglia exhibit the mildest lesions in experimental infections of poliomyelitis. This suggests the possibility that the nerve tissues are antagonistic to the virus. The adrenal, being one of the chromaffin tissues, may exert a similar influence. He describes the technic of its administration. Two c.c. of a 1 to 1,000 epinephrin solution should be injected intraspinally every four to six hours. "Before the first injection is given, a fairly large quantity of spinal fluid should be withdrawn, the quantity being in proportion to the pressure prevailing in the spinal canal. The subsequent injections should be made without regard to the presence or absence of spinal fluid." The injection is given with 2 c.c. of normal salt solution. If no spinal fluid is present, the epinephrin should be washed in with at least 5 or 6 c.c. of salt solution. "In cases in which the encephalitic symptoms are predominant, it should be used in inverse proportion to the exciting effect which the injections may produce." The injections should be continued for four or five days after all paralysis has disappeared, "or at least until no further reduction in the extent of the paralysis has taken place." Meltzer also advises the administration of oxygen. Absorbed by the blood, and combined with the lipoids of the nervous tissue, it may form an unfavorable environment for the anaerobic poliomyelitic organism.

Lewis,[90] Haas[91] and others report favorable results by the use of epinephrin chlorid intraspinally.

Hoyne and Cepelka[64] say that "some patients showed marked improvement in from one-half to one hour following the epinephrin injection." On several occasions a patient with a totally paralyzed arm would make a voluntary movement of the arm within an hour after receiving the epinephrin. Such marked improvement, however, was

89. Meltzer, S. J.: New York Med. Jour., 1916, **104,** 337.
90. Lewis, P. M.: Med. Rec., New York, 1916, **90,** 540.
91. Haas, S. V.: Med. Rec., New York, 1916, **90,** 425.

seldom permanent, but gradual improvement in some cases seemed to be more rapid."

Bass[92] describes the symptomatic treatment of poliomyelitis. Complete rest of the patient is essential, even if plaster-of-Paris must be used to immobilize. Pain and hyperesthesia are further treated by bromids and opiates. Acetyl salicylic acid may also be used. Local applications of heat are beneficial, though warm baths should not be resorted to, since they occasion unnecessary movements by the patients. The prevention of improper attitudes on the part of the extremities must be carefully watched. Sandbags, adhesive plaster and frames for the bedclothes may be used as preventives.

Charlton Wallace[93] divides the orthopedic treatment into three stages. These are (a) the acute stage, (b) the stage of apparent paralysis, (c) the stage of convalescence. The orthopedic treatment during the first stage is generally aimed at the relief of pain. This is accomplished by absolute rest and immobilization in plaster-of-Paris dressings, supplemented by careful, judicious nursing. Prolonged bed treatment is important in the second stage, lasting two or three months. Daily sponging with alcohol, and skin rubbing to avoid decubitus should not be forgotten. In the bulbospinal type, the patient should be placed on a mattress resting on a fracture board. All movements should be restricted, especially any attempt to sit up. Plaster-of-Paris bandages are indicated to prevent contractures of the feet. The stage of convalescence extends from the beginning of the ambulatory period to the end of the first year. During this time every effort should be made to maintain muscle balance and stimulate the paralyzed muscles. Daily massage and manipulation of the active muscles should be performed. Overbracing is preferable in the early months, after which it is gradually diminished. Electrotherapeutics may be of some value during the first year.

Lovett[94] and many other orthopedists warn against overfatigue. Lovett states that the convalescent patient is allowed up under supervision after two to three months. "Braces bear about the same relation to the treatment of poliomyelitis that crutches do to the treatment of fracture of the leg." If the patient cannot walk without braces, or if in walking, unnatural attitudes are assumed, some form of mechanically sound, light and properly fitted apparatus is advisable. Massage should not be overdone. Lovett emphasizes the importance of muscle training. This consists in having the patient execute special movements, and at the time of the effort, assisting him manually. By such

92. Bass, M. H.: Arch. Pediat., 1916, **33,** 611.
93. Wallace, Charlton: Arch. Pediat., 1916, **33,** 599.
94. Lovett, R. W.: Med. Rec., New York, 1916, **90,** 705.

practices, Lovett believes that new paths will be created to carry motion impulses and thus "reconnect a cerebral impulse with a peripheral muscular contraction." Even in the very chronic cases, muscle training is beneficial. Operative treatment should not be considered for two years.

Whitman[95] notes that muscle training is difficult of application in young children. He urges the better equipment and centralization of the clinics that deal with the after-treatment of poliomyelitis.

Ashley[96] says that muscle training should be in skilled hands. He considers massage very useful if not overdone. Parents may be instructed how to give it. "Electricity," according to Ashley, "is a good placebo when administered with a weak current. Its best and legitimate use will be to bring the patient for frequent regular examination, once in seven to ten days, in order that the physician may keep the child under observation and combat early any tendency to deformity."

Sayre[69] advises the use of strychnin during the convalescent stage. Local application of heat is also beneficial. Sayre notes that in regard to the use of electricity, the pendulum has swung from enthusiastic advocacy to absolute skepticism.

Frauenthal[97] is enthusiastic about the value of electricity. He begins its use when paralysis first appears. The high frequency current may be used along the spine for ten minutes every three hours for three days, during the acute stage. In the treatment of the paralysis, Frauenthal thinks that electricity maintains the tone and function of the muscles. "The least possible amount that will produce a contraction should be used." The slow wave sinusoidal current is the most perfect current to use, since it is best borne by young children. He applies sponge electrodes at the origin and insertion of the muscle needing stimulation. Frauenthal also advises light massage and baths while the patient is in quarantine.

Orthopedists generally emphasize the importance of rest and absolute quiet during the first two or three months, and the institution of orthopedic measures only to prevent deformity. During the second period (after the third or fourth month) they uniformly advise the use of light massage and muscle training, warning against carrying such measures to the point of fatigue. The value of electricity is considered very much overestimated. As a general thing, no operative measures are advisable during the first two years, but braces and splints

95. Whitman, R.: Med. Rec., New York, 1916, **90,** 1062.
96. Ashley, D. D.: New York Med. Jour., 1916, **104,** 725.
97. Frauenthal, Henry W.: New York Med. Jour., 1916, **104,** 1042; ibid., 1917, **105,** 247.

properly applied may prove useful.[98] Such measures to be successful must be constantly supervised.

Geyser[99] recommends electrotherapy, using it, not to cause contractions, but as a method of applying heat to affected muscles. When the functional response is slow, he advises the use of the sinusoidal current, and later the faradic current and muscle training.

Gaenslen[100] discusses the management of the stationary or paralytic stage. Treatment must be directed toward the maintenance of an equally distributed muscular balance. The factors which influence this balance cannot be controlled by massage and electricity alone. Gaenslen pleads for a careful study of these cases. Bad results are frequently due to neglect and to lack of cooperation on the part of parents.

Ryerson[101] discusses which patients can be improved by apparatus and which by operation. He censures the use of braces and repeated tenotomies in numerous cases in which muscles are paralyzed, resulting in an aggravated condition.

Gallie[102] describes the technic and reports good results with the operation of tendon fixation in deformities allowing poliomyelitis.

Sharpe[103] offers a preliminary report on a method for the restoration of function to paralyzed muscles. This he attempts by the "intradural anastomosis of a nerve root whose motor cells are active to a paralyzed nerve root." He selects active nerves whose function can be dispensed with. The duration since operation in three cases reported is not sufficiently long to speak of results.

Various operative measures for the correction of deformities following poliomyelitis are discussed by orthopedic surgeons.[104]

Bardine[105] and Wright[106] make earnest pleas for proper team work of hospitals and social agencies in the after-care of infantile paralysis. The problem is as much a social one as it is orthopedic.

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104. Am. Jour. Orthop. Surg., 1916, **14,** 381.
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106. Wight, L. O.: New York Med. Jour., 1916, **104,** 585.

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